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ANNALS
OF
OTOLOGY, RHINOLOGY
AND
LARYNGOLOGY

VOL. 64 SEPTEMBER, 1955 No. 3

LXV

PHYSIOLOGICAL OR SECRETORY SIALOGRAPHY

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AND

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ANN ARBOR, MICH.

Sialography is the roentgen visualization of the salivary glands after radiopaque substances have been injected into their ducts, and is thus comparable to contrast studies of other excretory organs. There have been conflicting reports as to what constitutes the normal sialogram, the best method for injection, the most satisfactory contrast material, and the quantity of contrast material required to obtain a sialogram of good technical quality.

Since the emptying of contrast material from different organs, as shown by postevacuation films of the colon and postfat feeding films of the gall bladder, has been of as much diagnostic significance as the filling of these organs with radiopaque substances, it was felt that similar studies of the salivary glands might be of value. Such a mechanism must be based on the reflex chemical stimulation of the salivary glands to empty the contrast material from their duct systems, and the mechanism is therefore considered a physiological procedure.

From the Departments of Radiology and Otolaryngology, University of Michigan Medical School, Ann Arbor.

This study was aided by the Frank D. McKay Research Fund in Otolaryngology.

With a view to standardizing the sialographic procedure and defining the characteristics of a normal sialogram, this paper presents a modification of sialography which is termed: Physiological or Secretory Sialography.

REVIEW OF THE LITERATURE

The first "radiogram" of the duct system of the parotid glands was made in 1904 by Charpy[1] following the injection of mercury into an anatomical specimen. Arcelin[2] applied this procedure clinically in 1913 in the case of a calculus in Wharton's canal, using a bismuth solution prior to radiography. In view of the irritating nature of heavy metal solutions and the lack of safe and satisfactory contrast media, the development of sialography as a diagnostic procedure was halted for a decade.

Sicard and Forestier,[3] in 1922, injected Lipiodol® into the epidural spaces of humans as a treatment for lumbago and observed that the oil was disseminated upward and downward in the epidural spinal canal. They tried injecting this material intrathecally and, finding that it was well tolerated by the meninges, reasoned that it might be a safe contrast medium with which to explore other systems of the body. In 1925 Carlsten,[4] Uslenghi,[5] and Barsony[6] described independently the procedure of sialography using iodized poppy-seed oil compound. Jacobovici, Poplitza, and Albu[7] in the following year described the use of sialography in three patients, demonstrating respectively a salivary calculus, a tumor of the parotid gland, and a tumor of the submaxillary gland. In 1928, a case of parotitis in which sialography was used as a diagnostic aid was reported by Keith.[8]

Rocchi,[9] in 1930, attempted to determine the amount of radiopaque material which would give the best roentgen visualization of a salivary gland. His clinical material covered a wide range of diseases. Subsequently, numerous reports on the clinical uses of sialography were published, with good correlation of clinical, roentgenographic, and pathologic material. Comprehensive reviews were published by Feuz[10] in 1935 and 1936, and by Blady and Hocker[11,12] in 1937 and 1939. Samuels[13] in 1950 and Ollenrenshaw and Rose[14] in 1952 attempted to evaluate the significance of acinar filling of the salivary glands.

ANATOMY

The major salivary glands are compound acinous or racemose glands of the serous type surrounded by connective tissue capsules. The parotid gland is roughly triangular and lies below and in front

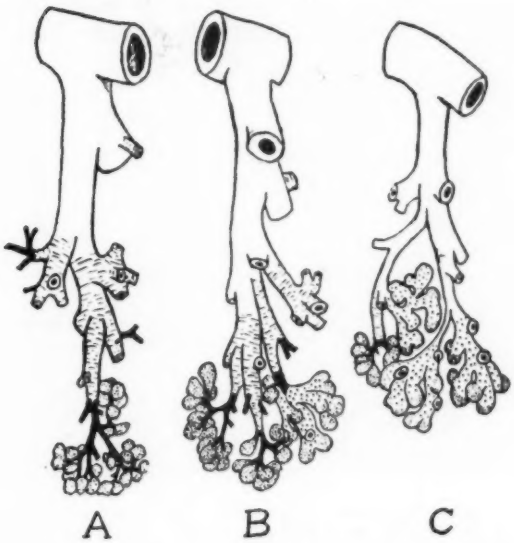

Fig. 1.—Simplified drawings of the duct system of salivary glands showing the various branches. A: Parotid gland, B: submaxillary gland, C: sublingual gland. (Reproduced from Bailey's Textbook of Histology, courtesy of Williams and Wilkins Co., Publishers.)

of the ear in the retromandibular fossa, extending from the zygomatic arch above to the angle of the mandible below. The parotid gland is of variable size and form but generally consists of a larger superficial lobe and a smaller deep lobe to which it is connected by an isthmus. The deep lobe or process retromandibularis is best seen in the frontal projection of the sialogram film. A separate portion of parotid tissue, the accessory lobe, is usually situated superior to the main duct and lies between the anterior border of the gland and the anterior border of the masseter muscle. Accessory lobes found were in 20 per cent of 76 anatomical specimens dissected by McCormack.[15]

The submaxillary gland is smaller than the parotid. It is partly lodged in the angle between the inner surface of the mandible and the muscular floor of the mouth, and projects downward into the digastric triangle of the neck.

The main ducts of the parotid glands (Stenson's ducts) and of the submaxillary glands (Wharton's ducts) are fibrous tubes of con-

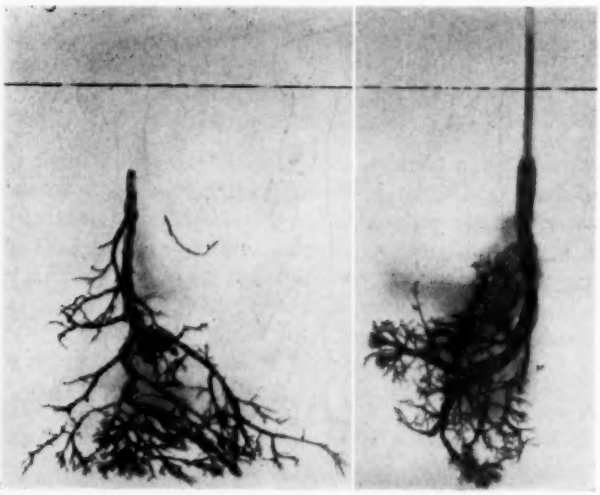

Fig. 2.—Casts of the duct system of the parotid and submaxillary glands prepared by injection of vanyl acetate into post mortem specimens. Right: parotid gland, left: submaxillary gland.

siderable length, approximately two inches (40-60 mm), and with a diameter of one-eighth inch (2-3 mm). The caliber of the ducts is considerably larger than the orifices which admit fine bristles, and for this reason the ducts may be considered reservoirs for saliva as well as ducts for its conveyance. The main ducts after division become the interlobar ducts, which then divide into the interlobular ducts, the various branches following connective tissue septa (Fig. 1). The interlobular ducts in turn become the intralobular ducts which are connected with the terminal alveoli through the intercalated ducts. This fine branching duct system can be appreciated readily in the normal sialogram. Casts of this duct system have been prepared by injection of vinyl acetate in acetone into post mortem salivary gland specimens, employing the technique that Tobin[16] and Liebow[17] devised to study the bronchial tree and its blood supply. The gland substance is dissolved in concentrated hydrochloric acid, leaving a colored plastic cast which can be mounted in clear plastic (Fig. 2).

HISTOLOGY

The secreting alveoli are made up primarily of serous cells, pyramidal in appearance, resting upon a basement membrane and sur-

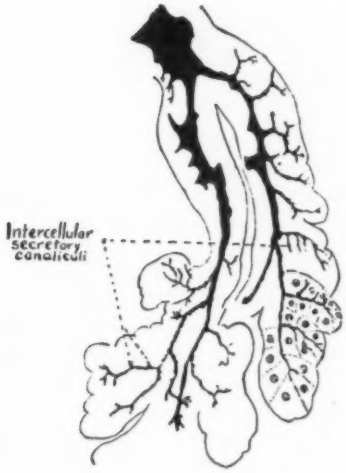

Fig. 3.—A drawing of the intercellular secretory and intracellular canaliculi demonstrated by the technique of Golgi. These canaliculi open into the acinar lumen and course between and penetrate into the cells. (Reproduced from Bailey's Textbook of Histology, courtesy of Williams and Wilkins Co., Publishers.)

rounding a fine narrow lumen. The appearance of these cells varies according to their particular phase of activity. In the resting phase the cells are swollen because the cytoplasm is filled with highly refractile zymogen granules which are the intracellular enzyme precursors. After active secretion, there is a diminution of zymogen granules and decrease in the size of the secretory cells.

By employing the technique of Golgi, fine intercellular secretory canaliculi may be seen in the secretory alveoli (Fig. 3). These canaliculi open into the lumina and extend between the cells, perhaps even to penetrate the cells themselves by delicate canals (intracellular secretory canaliculi) which are characteristic of secretory alveoli.[18]

STUDY OF FILLING

There would seem to be some controversy regarding the amount of contrast material necessary to produce a satisfactory sialogram. The amounts advocated by different investigators are listed in Table I and range from 0.5 cc to 6.0 cc. The maximum quantity tabulated is twelve times the minimum amount. If 2 cc of contrast material

is considered the mean quantity to be used, then one notes that there is a nearly equal number of authors suggesting quantities on either side of this arbitrary dividing line. Because of these conflicting reports, a careful review of the literature was undertaken to determine the ideal amount of material to be used. In addition, analyses of clinical data and experimental studies were made in order to answer two obviously fundamental questions: 1) What is the amount of contrast material necessary to obtain a sialogram of good technical quality in the normal gland? 2) What is the end point of injection, or how much filling of the duct system is necessary for an adequate study?

What is the amount of contrast material necessary to obtain a sialogram of good technical quality in the normal gland? The most obvious reason for the variance in the quantities tabulated is the type of patient studied. Most estimates were based on studies of diseased and enlarged glands rather than normal glands. The earliest documented studies of normal salivary (parotid) glands were made on post mortem material by Rocchi[9] in 1930. Four successive injections of 0.5, 1.0, 1.5, 2.0 cc of Lipiodol into isolated specimens were made and films were obtained after each injection. Rocchi concluded that the best roentgen visualization of the duct system was obtained after injection of 1.5 to 2.0 cc of Lipiodol.

Blady and Hocker,[11] in studying patients without evidence of salivary gland disease, divided their material to be injected into graduated doses and correlated the degree of filling that produced maximum visualization with the amount of pain produced by the injection. The arbitrary rule they advised was, "the total injection should be three to five times the amount producing discomfort and pain." A good sialogram in the average individual required between 1.0 and 1.75 cc, which was in agreement with Rocchi's work. They stated that less than 1.0 cc produced incomplete filling of the duct system.

In repeating these correlation studies in normal individuals, we have found that the amount of injected material required is one-half to one-third that advocated by Blady and Hocker, i.e., 0.5 cc to 1.0 cc (mean 0.8 cc). In other words, when the patient complains of definite pain, not just discomfort, the end point of the injection has been reached. The difference in our results lies perhaps in the method of injection. A closed system is employed as opposed to an open system of injection, which implies that the full amount of contrast material injected is retained in the system. To keep the system closed, injection is accomplished through a polyethylene catheter which is occluded when the procedure is finished. In the open method a blunt

Fig. 4.—Fractionated injection study of filling of the normal parotid duct system. 0.1 cc of Pantopaque fills the main cut.

edge needle is employed which is removed from the duct upon completion of the injection, allowing the contrast material to leak from the duct orifice while the films are being taken. This can be recognized easily in the reproductions of sialograms as collections or droplets of oil on the floor of the mouth or about the orifice of the duct. A sialogram to simulate the open method of injection is illustrated in Figure 8, the film taken after removal of the polyethylene catheter. Comparison with the other films in this study illustrates that the duct filling is less than the filling obtained with 0.5 cc of radiopaque material. Thus if the amount lost through leakage is estimated to be about 0.5 cc, the higher values of Blady and Hocker and other authors approach our figures.

What is the end point of injection, or how much filling of the duct system is required to render the study adequate? Figures 4 through 9 indicate the various degrees of filling correlated with the amount of contrast material used. With 0.1 cc of contrast medium

Fig. 5.—0.2 cc of Pantopaque shows filling of the interlobar ducts.

the main duct is visualized. With 0.5 cc the interlobar and interlobular ducts or secondary and tertiary branches are demonstrated; and the appearance is that of an American elm tree, stripped of its leaves (Figures 5 and 6). After injecting between 0.75 cc and 1.0 cc, the interlobular and intercalary ducts are visualized plus minimal acinar filling which represents ideal filling (Figure 7). More extensive acinar and alveolar filling results when 1.0 to 2.0 cc of contrast material is injected which may produce partial obscuration of the secondary ducts and their ramifications (Figure 9). Thus the previously mentioned elm tree appears to be in full leaf with the branches obscured.

Diffusion of contrast material into the gland substance in sialography has long been recorded. In the German literature, the roentgen picture of diffusion has been termed "wolkenartigen schatten" (cloud-like shadow) and "apfelblueten" (apple blossom). In the English literature, Samuels[13] described this phenomenon, believing it to be a new finding of uncertain origin. He described the "sialo-

Fig. 6.—0.5 cc of pantopaque demonstrates the interlobular and intralobular ducts. At this point the pressure of injection is noted by the patient.

acinar reflux" and questioned whether this was a normal finding due to overinjection or the increased permeability of the ducts as a result of infection. Ollenrenshaw and Rose[14] in reviewing their material felt that the "sialo-acinar reflux" was due to the filling of the acini themselves and did not represent an increased permeability of the ducts. They reasoned that patent acini could be filled, provided the amount and pressure of the injection medium were adequate. To prove this point they made sialograms of normal patients, using manometric equipment to control the pressure of injection. It was demonstrated that 2.5 cc had to be injected before any acinar filling occurred. At this point the manometric reading was 400 mm Hg., which the authors stated "sounds frighteningly high but is only that of a firm finger applied to a syringe." Furthermore, these authors concluded that failure to fill the acinar structures indicated that the injection had not been carried to completion. With the realization that it is impossible to control all injections routinely by manometric

Fig. 7.—0.8 cc of Pantopaque shows filling of intercalated ducts and minimal fillng of the acini, resulting in partial obscuration of tertiary and terminal ducts. At this point, the onset of pain was noted by the patient, indicating the end point of injection.

pressures, they advocated deliberate overinjection to obtain films of greater diagnostic value.

Our experience is contrary to that of Ollenrenshaw and Rose, since we have found that overinjection of salivary glands is painful, may be harmful to the glandular parenchyma, and depreciates the diagnostic value of the sialograms. The capacity of the duct system of the average parotid gland, including the acini, in health or disease, rarely exceeds 2 cc. The submaxillary gland has a slightly smaller capacity. If more than 2 cc of contrast material are injected, the capacity of the gland is exceeded. As a result, the contrast material will leak around the catheter at the duct orifice and will be observed as collections of contrast material on the floor of the mouth. Also, the contrast material will fill the lumina of the acini and even enter the cells themselves via the intercanalicular and intracanalicular chan-

nels, causing their distention and eventual rupture. Clinically, the gland may be distended to about three times its normal size.

It might be argued that if the gland enlarges immediately after injection it will return to normal size in two or three days, indicating that no significant damage has been done. Epsteen and Bendix,[31] in evaluating the effects of various contrast media, injected 1.5 cc to 2.5 cc into salivary glands of dogs. Two weeks after the initial swelling subsided, the glands were excised and examined histologically. The microscopic sections showed areas of necrosis and foreign-body reaction. These experiments are borne out in one unfortunate case in our series in which the parotid gland was overinjected with 5 cc of contrast material.

REPORT OF A CASE

C. F., a 15 year old colored male, noted left parotid enlargement six weeks prior to admission to the University of Michigan Hospital. The swelling had increased progressively without associated pain or other symptoms. A previous sialogram had been made using the open method of injection. It was reported that 5 cc of iodochloral was used, but the exact amount injected could not be estimated, since a considerable quantity was lost through leakage. The procedure was extremely painful to the patient. The films showed a bizarre accumulation of contrast material which obscured the entire duct system and prevented any diagnosis of an infectious or neoplastic process (Figure 10). This was interpreted as representing an overinjected gland, with contrast material rupturing the acinar structure and dissecting along the connective tissue septa. Subsequently, the glandular swelling increased and became tender, establishing a diagnosis of acute suppurative parotitis. Hot compresses and antibiotics were used, but the condition progressed to suppuration and surgical intervention was advised. At this time, fourteen days after sialography, an x-ray examination showed definite evidence of residual contrast material, though the amount was less than that shown in the original films. At operation the tense superficial portion of the gland was excised. After removal, the specimen showed no gross evidence of neoplasm. The normal parenchymal lobular pattern was recognized and in several areas small amounts of oily, mucinous material were noted, which seemed to indicate points of rupture of the ducts during sialography.

The histologic sections of this gland showed large and small ducts surrounded by relatively heavy lymphocytic infiltrations containing occasional multinucleated giant cells (Figure 11). The pathologic diagnosis was active chronic inflammation. No evidence of neoplasm or specific chronic infectious granuloma was observed. Many vacuolated areas were seen which were thought to represent droplets of oily contrast material in the gland parenchyma. The patient's postoperative recovery was uneventful.

There are two basic principles which must be respected if relatively painless and satisfactory sialography is to be accomplished:

1. No preconceived or precalculated amount of contrast material should be injected into a salivary gland. The amount used should be governed by the production of pain. The end point of injection is the moment when definite pain occurs during utilization of a closed system. Perhaps the reason Blady and Hocker selected

Fig. 8.—Simulation of the open method of injection. Removal of the polyethylene catheter upon completion of injection shows loss of contrast material at the duct orifice. Comparison with Figure 6 illustrates that the duct filling is less than that obtained with 0.5 cc of Pantopaque.

an amount three to five times the amount at the threshold of pain was to compensate for the leakage of contrast material in their open system of injection. The pain produced is probably due to sudden capsular distention and is akin to salivary gland pain in acute inflammatory or obstructive processes. In benign neoplastic disease, where capsular distention is gradual, no pain occurs. Therefore the injection should be slow and controlled. As stated previously, the amount which can be injected into normal glands may vary between 0.5 cc and 1.0 cc. In diseased states there is a wider range in the quantity which must be used to obtain a good sialogram. In infectious and obstructive conditions producing dilation of the duct system, quantities up to 2.0 cc have been used. In neoplastic disease the required quantity is usually that required for a normal gland, or even less since the gland parenchyma may be replaced by the neoplasm.

2. The ideally injected gland shows filling of intercalary ducts and minimal delineation of the acini in the roentgenogram. Over-

Fig. 9.—A sialogram of the submaxillary gland. Injection of 1.0 cc of Pantopaque into the submaxillary gland demonstrates the duct system to good advantage. Clouding of the gland substance is evidence of acinar filling.

injection will lead to rupture of acinar cells and obscuration of the ducts. Quantities greater than 2 cc, even in disease, are rarely necessary for diagnostic purposes and are not well tolerated by the patient. The cumulus-cloud effect of extensive acinar filling is as undesirable in sialography as is alveolar filling in bronchography (compare Figures 10 and 12).

STUDY OF PHYSIOLOGICAL EMPTYING

Radiographic study of the emptying of the contrast material from the salivary glands has been neglected. The misconception perpetuated by repetition in the literature[10-12,14,19] is that "Lipiodol empties from the gland in one to three days in the normal. When greater quantities of contrast medium are used or when repeated studies are done, a residual can be seen on films five to fifteen days later." These

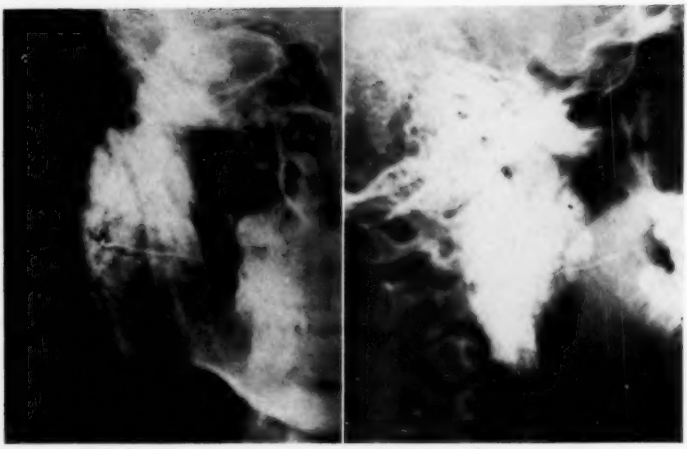

Fig. 10.—A. An overinjected sialogram. A-P projection of the parotid gland following injection of 5 cc of iodochloral by the open method. B. Overinjected sialogram on the same patient. The lateral projection shows the bizarre appearance of the accumulated contrast material which has obscured the entire duct system.

statements appear to be incompatible with the physiological fact that the average salivary gland (parotid or submaxillary) secretes 25-50 cc of saliva per hour by continuous stimulation, as in mastication of food. If stimulation of the gland can produce such a profuse secretion, it would appear unlikely that it would be unable to wash a few cubic centimeters of contrast material from its duct system.

A review of the basic physiology of secretion of the salivary glands was undertaken to determine if any information so derived could be applied to sialography. In the normal life of the animal, the secretion of saliva is initiated reflexly either by inherent or by acquired (conditioned) stimulation. In the first instance materials placed in the mouth call forth, after a short latent period (2 to 3 seconds), a secretion of saliva which varies in quantity and quality with the physical nature of the stimulating substance. The effects which sensations of taste produce on secretion of saliva are well known. Usually the most palatable substances are the most potent salivary stimulants; however, substances unpleasant to the taste, such as strong organic acids, are also potent sialogogues.

In applying the above physiological phenomena to sialography, the patient is instructed to suck on a lemon, as a source of citric acid,

for one minute. Stimulants such as minted gum, candy, or diluted vinegar also may be used effectively. Thus, as the gland is stimulated the contrast material is washed from the duct system, after which the patient rinses his mouth to remove the radiopaque substance prior to postevacuation roentgenography. In a study of more than twenty-five normal glands, all have emptied within five minutes if properly injected. Studies made with rapid changing of films show that the gland empties completely within the first minutes after lemon stimulation. Occasionally, if some acinar filling occurs, a faint cloud of contrast material outlines the gland in the five-minute poststimulation film. However, this cloud is usually absent on the one hour film and has disappeared on the twenty-four hour poststimulation film.

Therefore, it is concluded that retention of contrast material in a normal gland for more than five minutes after stimulation indicates an overinjected gland. Allowing for occasional acinar filling, no residual opacity should be seen on the twenty-four hour poststimulation film if the normal gland has been properly injected. If contrast material is retained for days or weeks in a normal salivary gland, it is safe to assume that the gland has been overinjected and the acinar cells have been ruptured. The degree of cellular destruction determines the amount of secretory function remaining which in turn determines the length of time residual solution is retained in the gland. As Epsteen and Bendix[31] have demonstrated in their experimental studies, Lipiodol which is retained in salivary glands will lead to pathological changes. Thus it is essential to insure proper injection of the gland, the objective test of proper injection resting on the ability of the stimulated gland to empty the contrast medium completely within five minutes.

This technique of physiological or secretory sialography has been applied to more than one hundred sialograms, and the retention of contrast material in these cases has proved to be of diagnostic value. The retention pattern is characteristic in different entities. The results of these studies will be published in the near future.

CONTRAST MEDIA

According to Neustaedter et al,[32] the ideal radiopaque mixture must possess at least four important features: 1) Proper viscosity, 2) capability of rapid absorption and excretion, 3) freedom from injurious effects on the tissues, 4) sufficient radiopacity to render good delineation of structures under examination. Most of the available literature deals in detail with unfavorable tissue reactions following injection of various solutions into different body cavities.

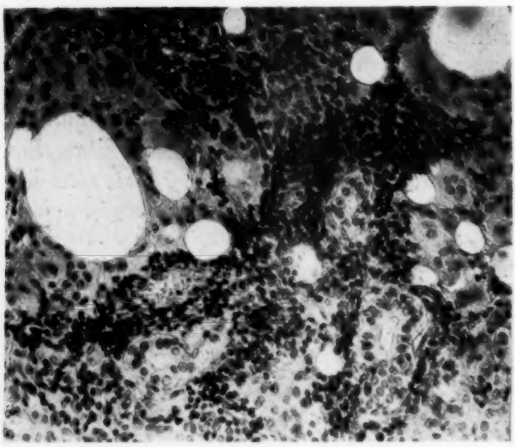

Fig. 11.—The microscopic section of the overinjected parotid gland shows heavy lymphocytic infiltration about the small ducts. The vacuolated areas represent droplets of oily contrast material in the gland parenchyma.

Recently, Epsteen and Bendix[31] have studied the effects of injection of various oily contrast substances in the salivary glands of dogs.

In the present study we have employed the following contrast materials:

Lipiodol—40 per cent in stable iodide compound in poppy-seed oil.

Iodochloral—27 per cent iodine in organic combination in a refined peanut oil.

Pantopaque®—30 per cent iodine in an iodized, phenyl-substituted fatty acid derivative: ethyl iodophenylundecylate.

The use of Pantopaque is preferred because it has been found to fulfill the above criteria most satisfactorily. It is of lower viscosity than the other materials and thus provides for ease and control of injection. The low surface tension of Pantopaque permits ready diffusibility and greater penetration of the finer ducts under low injection pressures. In diseased glands, Pantopaque tends to intermix with retained secretions rather than to form droplets and thereby create confusing shadows. In addition, its lower viscosity means less

Fig. 12.—A normal parotid and submaxillary sialogram. Injection of each gland was made consecutively to determine the character of an extrinsic mass in the neck. Both the parotid and submaxillary areas were involved. Note the calcified lymph nodes on the opposite side.

surface resistance and increased facility of excretion. This is of considerable importance in disease states where the secretory function of the gland is impaired.

The nonirritability and radiopacity of the above listed contrast materials are about equal in sialography. The production of pain as well as the pathological changes are due to overinjection and retention. Both poppyseed oil (the vehicle of Lipiodol) and peanut oil (the vehicle of iodochloral) have been shown to produce marked foreign-body reaction, obliteration of ducts, round-cell infiltration, and areas of focal necrosis after injection into salivary glands of dogs. If the gland is properly injected, however, and upon immediate stimulation empties all of the contrast medium, it is unlikely that such unfavorable reactions will occur in normal glands.

In pathological conditions, retention of contrast media will occur even after stimulation, and thus the problem of histologic changes due to this retention deserves consideration. In chronic inflammatory and obstructive diseases due to calculi, the use of aqueous contrast material is being explored. Urokon® sodium is more irritating than the oily materials; however, the unpleasant effects are transitory and

the Urokon is absorbed rapidly by the acini in a matter of minutes. In neoplastic disease, retention of the contrast medium is of little importance, since most of the salivary gland is usually resected when the tumor is removed. Also the secretory phase of sialography is so important in the differential diagnosis of palpable masses in and about the salivary glands that the use of aqueous contrast material is contraindicated. No untoward effects or complications have been observed after using Pantopaque.

TECHNIQUE OF INJECTION

Many physicians feel that sialography is too difficult and time-consuming to be useful in everyday practice. As is true of many techniques, however, once it is mastered it becomes simple and efficient. The estimated time required for the average sialogram under favorable conditions is twenty minutes. The equipment consists of a set of Williams olive-tipped lacrimal probes, Bowman (plain-tipped) lacrimal probes, a 2-cc hypodermic syringe, and a polyethylene catheter #60, which is equivalent to a 21-gauge hypodermic needle. The patient is usually placed in the sitting position and a source of good illumination should be available, preferably a directly or indirectly illuminated head mirror.

The location of the orifice of Stenson's duct is in the papilla of the buccal mucous membrane opposite the second molar tooth. The orifice of Wharton's duct is readily found on the floor of the mouth on either side of the midline at the base of the frenulum of the tongue. Topical application of a solution of 10 per cent cocaine hydrochloride to the duct orifice not only reduces the discomfort associated with dilation, but makes the orifice visible by stimulating salivary flow. Aseptic technique is used throughout the procedure.

A punctate director is inserted into the mouth of Stenson's duct, after which dilation is accomplished with the olive-tipped probes. The duct should be probed through its entire length with the smallest probe (size #1) to ascertain the patency and character of the lumen. This implies insertion of the probe into the hilum of the gland which lies beyond the anterior border of the masseter muscle. For purposes of dilation the probes are inserted up to the masseter border, using sizes #1 through #4. This procedure is virtually painless so long as the probes are directed along the natural course of Stenson's duct. A polyethylene catheter (P.E. #60), sterilized in 1:500 aqueous benzalkonium chloride for at least 24 hours, is then inserted into the duct for a distance of 3 to 4 cm. This is best accomplished with the aid of a fine rigid wire stylet onto which an 8 to 10 cm section of polyethylene tubing is threaded. Precautions should be taken to avoid

TABLE I.—AMOUNTS OF LIPIODOL USED IN SIALOGRAPHY BY VARIOUS INVESTIGATORS

REFERENCE NUMBER	AUTHOR	CUBIC CENTIMETERS
4	Carlsten (1925)	5.0
7	Jacobovici et al (1926)	4.0 (20% iodopine)
9	Rocchi (1930)	1.5 - 2.0
10	Feuz (1932)	2.0 - 4.0
11	Blady and Hocker (1938)	1.0 - 1.75
13	Samuels (1950)	0.5 - 1.0
14	Ollenrenshaw and Rose (1951)	2.5 - 3.0
19	Rose (1950)	1.5 - 2.0
20	Berraud (1931)	2.0 - 4.0
21	Payne (1931)	0.5 - 1.0
22	Hobbs et al (1932)	1.0 - 1.5
23	Jacobovici and Jianu (1933)	4.0 - 6.0
24	Simon (1934)	2.0 - 5.0
25	Grasso (1934)	1.5 - 2.0
26	Hare (1935)	0.5 - 1.0
27	Kimm et al (1935)	1.0 - 1.5
28	Schulz and Weisberger (1950)	1.0 - 1.5
29	Putney (1950)	1.0 - 1.75
30	Antoine et al (1952)	2.5

protrusion of the stylet beyond the open end of the polyethylene catheter. The stylet should be of curved contour similar to that of the lacrimal probes. Traumatization of the duct, which occurred quite frequently when a blunt needle was used, rarely occurs when a polyethylene catheter is employed. The submaxillary gland is dilated in a similar manner with the Bowman (plain-tipped) lacrimal probes. Because of the hypermobility of the delicate sublingual papilla, the olive-tipped probe is apt to cause intussusception and edema about the salivary caruncle and should not be used.

The injection should be carried out slowly. In the majority of patients, a definite pressure sensation occurs when 0.25 cc to 0.5 cc has been injected into either the parotid or submaxillary gland. Continued injection is accomplished slowly until the patient signals that definite pain is present. This is the end point of the procedure and indicates early acinar filling and beginning capsular distention. The total amount of contrast material in pathological conditions varies between 0.5 and 1.5 cc and rarely exceeds 2.0 cc, as previously stated.

Upon completion of injection, the open end of the catheter is plugged with a wooden stopper (a toothpick).

Roentgenographs are then taken of the injection phase. The catheter is removed and the salivary gland is stimulated by allowing the patient to suck on a slice of lemon, minted candy, or chewing gum for one minute. Poststimulation films are taken after the mouth has been rinsed.

RADIOGRAPHIC TECHNIQUE

Films are taken with the patient in the horizontal position on a Potter-Bucky table, using the technique for routine skull examination. The following films are taken for both parotid and submaxillary sialograms: Preliminary films: P-A and lateral; injection films: P-A and lateral; poststimulation films: P-A and lateral.

Poststimulation films are made after five minutes, and if retention is shown, again after one hour and twenty-four hours. For the P-A projection the Granger projection is employed. The use of stereoscopic films during the injection phase is preferred.

PURPOSES OF SIALOGRAPHY

1. To determine if a mass in the parotid or submaxillary area is intrinsic or extrinsic, inflammatory or neoplastic, encapsulated or invasive.

2. To aid in the planning of surgery in the involved area, particularly in neoplastic disease where the relationship of the tumor to important contiguous structures is of prime significance.

3. To demonstrate the presence of calculi, particularly the radiolucent variety, and to determine their position in the gland or duct.

4. To confirm the clinical diagnosis of sialectasis, fistulae, or strictures.

5. To help select a biopsy site, if indicated.

The only contraindication to sialography is acute inflammation, as in mumps, postoperative (dehydration) parotitis, etc.

SUMMARY

A review of the literature on sialography shows that radiographic study of the emptying of contrast material from salivary glands has been neglected, and that therefore certain misconceptions have been perpetuated. This article presents a new modification of sialography

termed "physiological or secretory sialography." The technique of injection and radiographic filming requires about twenty minutes. The contrast material of choice is Pantopaque, because of its low viscosity, diffusibility, and facility of excretion. The important features of the technique are: The use of a polyethylene catheter, a closed system of injection, and physiological reflex stimulation of the salivary gland to evacuate the contrast material. Stimulants such as lemon juice, minted gum, candy, or diluted vinegar have been used to obtain a profuse salivary flow. In obtaining a sialogram of good technical quality and with the least discomfort to the patient, the following principles are important:

1. No preconceived or precalculated amount of contrast material should be injected into the salivary gland. The amount used should be limited by the production of pain. As a general rule, the amount in health or disease should not exceed 2 cc; usually it is between 0.5 and 1.5 cc.

2. The well-injected gland shows by x-ray a filling of intercalary ducts and minimal demonstration of acini.

The retention of contrast material in a normal gland for more than five minutes after stimulation indicates the injection of too much solution. Overinjection is extremely painful and may result in pathological changes in the gland parenchyma, thus decreasing the diagnostic value of the study.

Sialographic study is indicated in cases of suspected neoplasm, calculi, sialectasis, fistulae, or strictures; it is useful also in selecting a site for biopsy and in planning surgical operations in the involved area.

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LXVI

OSSIFYING FIBROMA OF THE PARANASAL SINUSES

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Ossifying fibroma, a variant of the intraosseous form of fibroma, has been described as involving various bones of the face and skull.[1-15]

A thorough study of the literature of the past half century has revealed that most of these tumors have been reported under different names. Because of the confused state which existed regarding the differentiation between certain types of sarcoma, ossifying fibromas have been referred to as "sarcoma," "sarcoma psammosum," "sarcoma osteogeneticum." More commonly, however, the term "psammoma" was applied.

The absence of nuclear monstrosities, mitotic figures and protoplasmic anomalies has induced most recent authors to consider these growths as benign tumors and designate them as fibromas.

Goegl[8] proposes the comprehensive term psammo-osteoid-fibroma. American and British authors generally report them as calcifying or ossifying fibromas.

The relationship between ossifying fibroma and monostotic fibrous dysplasia is very significant.

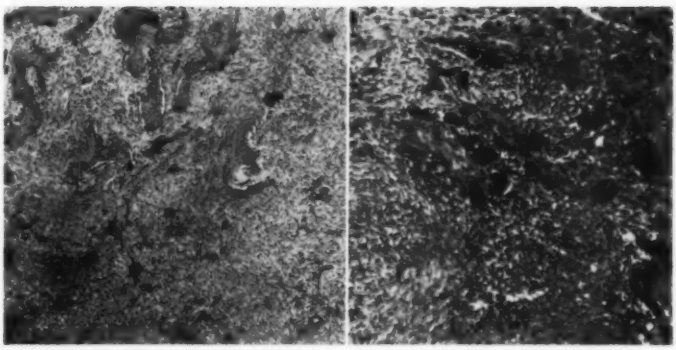
"Fibrous dysplasia of the bone" is the name applied by Lichtenstein[16-17] to a polyostotic or monostotic condition apparently due to a perverted activity of the specific bone-forming mesenchyme. It is most frequently localized in the femur, tibia, rib, maxilla and mandible and it occurs generally in older children and young adults. Microscopically, spicules of osteoid tissue or calcified bone trabeculae are embedded in a moderately or markedly vascular fibroblastic tissue; there are marked variations of the cellularity of the fibrous tissue from area to area with occasional hyaline cartilagenous tissue, hemorrhages and cyst formations. Among the extraskeletal aspects of the condition, abnormal pigmentation of the skin, premature sexual development in females and signs of hyperthyroidism have been described in very severe conditions.

According to Schlumberger,[18] the monostotic type of fibrous dysplasia represents an abnormal response to injury of the bone as a disturbance of the normal reparative process following trauma. It is generally agreed by most authors (Lichtenstein,[19] Mallory,[20] Schlumberger,[4-18] Szanto[21]), based on clinicopathologic grounds, that ossifying fibroma is a variant of monostotic fibrous dysplasia. According to Szanto[21] "the only difference that exists is that in ossifying fibroma the spherical islets of osteoid tissue predominate over the bony spicules so characteristic of monostotic fibrous dysplasia." Stages intermediate between the trabecular and spherical type of bone formation have been described.[18] Smith and Zavaleta[5] consider monostotic fibrous dysplasia of cranial or facial bones as synonymous with ossifying fibroma.

Clinically, Brunner[22] maintains that in fibrous dysplasia the sinuses are entirely filled with newly formed tissue; that in many instances dysplasia of the skull prevents the development of the paranasal sinuses by producing pathologic tissue which takes the place destined for the respective sinuses and therefore the radical removal of the pathologic tissue is impossible unless the facial bones harboring the involved sinuses are sacrificed to a great extent; that as a rule several facial bones are involved in fibrous dysplasia and there is a tendency to fill up the surgical defect with new bone.

Further distinctions should be made between ossifying fibroma and osteoma.

As there is a great variability in the amount of fibrous and osseous tissue in ossifying fibromas, Phemister and Grimson[3] called "fibrous osteoma" the more mature type with extensive ossification, and suggested the term "osteo-fibroma or ossifying fibroma" for the group in which fibrous tissue and immature bone predominate. Geschichter and Copeland[23] expressed the idea of the evolvement of ossifying fibromas into osteomas. An evaluation of twenty ossifying fibromas and twenty osteomas of the cranial and facial bones by Smith and Zavaleta[5] demonstrated a parallel between the age of the patient and the maturity of the tumor. Ossifying fibroma progresses from young to intermediate to mature forms. And it was also observed that with advancing age there was an apparent evolution from the mature ossifying fibroma to a transitory osteoma and finally the mature form of osteoma. According to these authors "it is only natural to reason that since the normal end result of fetal membranous ossification is mature bone, the expected result of a mass of similar fibrous ossifying tissue is an osteoma."



A B

Fig. 1.

The purpose of this paper is to report four cases of ossifying fibromas involving the paranasal sinuses and to correlate the clinical, radiological, surgical and pathological data in relation to the establishment of a preoperative identification of this clinico-pathological entity.

REPORT OF CASES

CASE 1. E. V., a white woman aged 28, was first seen in the Clinic, Research and Educational Hospital on Dec. 18, 1950, complaining of frontal headache, pain over the right cheek and diplopia.

The onset of complaints started three years before during which time she had been treated for sinusitis. Her headache varied as to intensity and frequency, but mainly localized to the right supraorbital area. The right infraorbital area was tender and reddened especially during the evenings. The patient delivered three months before and during pregnancy her right eye was intermittently closed; diplopia was present when looking up and to the right. Needle biopsy done elsewhere was negative.

Physical examination revealed a well-developed and well-nourished female, with moderate ptosis of the right lid and slight fullness of the right cheek. Examination of the nose showed some mucopurulent secretion in the right middle meatus and redness of the nasal mucosa on the same side. The right infraorbital ridge was irregular to palpation. The ears and rhinopharynx were normal.

Transillumination of the sinuses disclosed a very dense right maxillary sinus whereas the other sinuses were clear. Ophthalmologic consultation revealed only ptosis of the right eye with a small palpebral fissure.

The x-ray examination of the paranasal sinuses demonstrated a diffuse opacity of the right maxillary sinus with irregular destruction of its superior and upper lateral walls; there was slight clouding of the right frontal and ethmoid sinuses. Films of the skull were normal with no evidence of invasion of the base of the skull. Blood chemistry and urinalysis were normal.

The patient was admitted to the Research and Educational Hospital of the University of Illinois. A Caldwell-Luc approach was used to obtain a biopsy from the right maxillary antrum. The anterior wall of the antrum was intact. The sinus was found to be occupied by a very firm, granular mass which could not be separated from the overlying bone. A small amount of free space existed medially and inferiorly to the mass.

Microscopically, there were numerous calcified and noncalcified bony trabeculae embedded in moderately cellular and fairly vascular connective tissue stroma. The bony trabeculae contained relatively few osteocytes. Along the bony trabeculae, rows of osteoblasts were seen (Fig. 1A). In addition there were sections which revealed a more cellular and vascular stroma. The tumor cells were spindle-shaped and elongated. They were arranged in bundles. The nuclei were fairly uniform in size and shape and in staining qualities. Throughout the section numerous broad bony trabeculae were seen containing mature osteocytes. Along the surface of some of the bony trabeculae numerous osteoblasts were noted. In some areas numerous multinucleated giant cells were seen. They contained between ten and twenty nuclei. Some of them contained vacuoles and looked like osteoclasts. Many of them appeared to be independent from bone trabeculae. Others, however, were around small trabeculae which showed extensive bony absorption (Fig. 1B).

The excision of the tumor was performed through a Moure incision. The anterior wall and a portion of the lateral wall of the maxilla was removed. The mass had broken through the infraorbital plate and this was removed; the overlying periosteum was intact. The mass extended to the nasal process of the maxilla which was partially removed. The nasal wall and the floor of the antrum were not involved.

Recovery was uneventful and the patient was discharged from the hospital on the seventh postoperative day. The incision healed well with little depression of the right cheek. Five series of x-ray films of the sinuses and skull revealed the surgical defect in the right maxillary region without evidence of recurrence four years postoperatively.

In this case there was an ossifying fibroma of the right maxillary antrum with clinical symptoms of frontal headache, pain and redness over the right cheek, ptosis of the right eye and recurrent diplopia.

At operation a firm, granular mass was found involving the facial, lateral and superior walls of the antrum. No recurrence was seen four years after surgical removal.

Finally the histopathological picture showed not only areas with a coarse network of bony trabeculae embedded in fibrous tissue, but also areas of interspersed lamellae of bone and areas with multinucleated giant cells of osteoclastic type. Goegl,[8] who reported one case with a similar polymorphous appearance, maintains that these tumors undergo a certain transformation and the histologic slide represents only the picture of the moment. The bony islets in particular are subject, as far as stability, form and size are concerned, to the structural changes (osteogenesis and osteoclasis) and growth of these neoplasms. On the other hand we have already reported the above possibility of evolution of these "mature" ossifying fibromas (moderately dense fibrous tissue, trabeculae of calcified bone, prominent osteoblast and osteoclast layering around bone) into osteomas according to Smith and Zavaleta.[5]

Fig. 2.

CASE 2. T. T., an eight year old white male, was first seen at the ear, nose and throat clinic, Research and Educational Hospital on October 27, 1952, because of a painless swelling of the left cheek. The parent noticed the swelling only two months before when the child was hit on the left side of the face by a ball. The patient complained of frontal headache of various intensity and frequency and not associated with ocular symptoms.

Physical examination revealed a well-developed, alert and co-operative male with a smooth swelling involving the left maxilla, including the alveolar ridge. Examination of the nose did not reveal clinical evidence of sinus infection; the rhinopharynx and ears were within normal limits.

Transillumination of the sinuses disclosed an increased density of the left antrum; the other sinuses were clear.

The x-ray examination of the skull, paranasal sinuses and jaws revealed a discrete bony mass which occupied most of the maxilla and had invaded the left maxillary antrum, appearing radiologically as a benign osteoma. X-rays of the long bones and the blood chemistry were within normal limits.

On Nov. 25, 1952, through a Caldwell-Luc approach, a biopsy was taken. The canine fossa showed an area of destruction of about one centimeter; the left antrum was filled with a soft reddish mass.

Microscopically the sections revealed a very cellular tissue. The cells were young fibroblasts arranged in interlacing bundles. In several areas there were actively growing sheets of cells with indistinct cytoplasm and large oval nuclei, with clumping of chromatin. A few multinucleated giant cells were seen. Scattered throughout the section were numerous islands of bone. Many of these islets were eosinophilic containing young osteocytes. Along these islands of bone numerous osteoblasts were seen. Some of the islets were calcified.

On Dec. 18, 1952, the tumor was removed through a sublabial incision. It showed a fair amount of encapsulation along the orbital plate, the medial wall and the floor, but infiltrated into the lateral aspect of the maxilla and into the malar bone. The main body of the tumor was eviscerated. It was necessary to curette a considerable portion of bone overlying the tooth buds and the superior alveolar nerves and vessels. The lateral aspect of the maxilla was curetted free of tumor which extended into the malar bone up to the frontal and zygomatic processes leaving a thin shell of the malar bone.

Recovery was uneventful and the patient was discharged from the hospital on the fifth postoperative day. He has been under observation during the past two years. He had four series of films of the sinuses and maxilla; they revealed no evidence of recurrence. An x-ray taken two years after the surgical removal shows a normal maxilla and a clear left antrum.

This was a case of ossifying fibroma of the left maxilla.

Clinical symptoms were a painless, smooth swelling of the left side of the face. It is well known that both benign and malignant tumors may remain unsuspected in the antrum for a long time until the expansion of the bony walls of the sinuses calls attention to their presence.

Removal was done through a sublabial incision with excision of the mass and curetment of the bones involved. In 1937, Phemister and Grimson[3] maintained that in view of the benign nature of the lesion, massive resection with its accompanying disfigurement and high mortality is unjustified; they suggested that the tumor be surgically removed as thoroughly as possible without too much of a destruction of the jawbone. Though excision and curetment were employed there is no evidence of recurrence after two years. It is recognized that pneumatic spaces do not develop in pathologic bone; in this case no difference could be elicited between the antra at the time of the first visit and no deviation from normal development could be shown two years after surgical removal of the tumor.

Histologically, it was a young-to-intermediate type of ossifying fibroma (dense fibrous stroma with actively growing cells, presence of osteoid tissue and islands of bone, giant cells separated from bone).

CASE 3. R. I., a seven year old white male, was referred by an ophthalmologist to the ear, nose and throat clinic, Research and Educational Hospital, on Nov. 20, 1953, because of an upward displacement of the left eye of two weeks' duration.

Physical examination revealed a well-developed boy with an obviously proptosed left eye and fullness of the left cheek. Examination of the nose revealed an obstruction of the left nasal cavity by an enlargement of the inferior turbinate and medial displacement of the left lateral nasal wall to such an extent as to displace the septum to the right. However, no tumor tissue was visible. The remaining physical examination was essentially normal.

Transillumination disclosed a very dense left antrum; the other sinuses were clear.

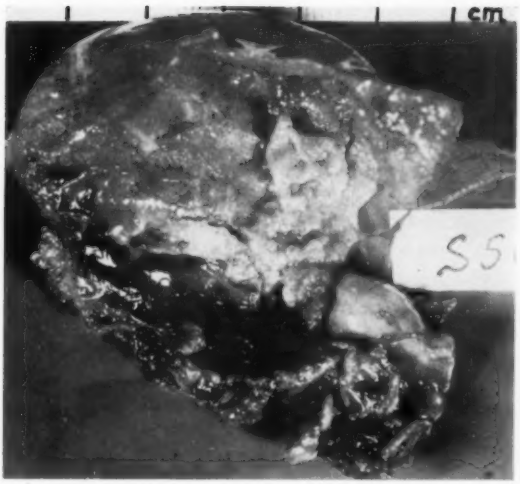


Fig. 3.

The x-ray examination of the paranasal sinuses revealed a density of the left maxillary, ethmoid and sphenoid sinuses; the left infraorbital plate was displaced upward while the lateral and medial walls of the left antrum were displaced respectively laterally and medially (Fig. 2).

The chest film was normal as well as the bone survey for metastasis or metastatic involvement of any kind. Complete blood study and urinalysis were within normal limits.

On Dec. 3, 1953, the left maxillary sinus was explored through a Caldwell-Luc approach. The anterior wall of the maxilla was found to be very thin and the antrum was occupied by a cyst whose walls were from one-eighth to one-fourth of an inch in thickness. Frozen section revealed a cellular osseous type of tumor consistent with ossifying fibroma. Therefore, the tumor was bluntly dissected and found to be expanding the infraorbital, lateral and medial walls of the antrum, but not actually invading them, as the cyst was easily freed from the mucosa of the antrum (Fig. 3).

Microscopically, the cystic wall was lined by respiratory epithelium; underlying this is a layer of edematous connective tissue infiltrated with chronic inflammatory cells; beneath this is a thin layer of bony islets surrounded by fibrous tissue infiltrated with chronic inflammatory cells. Other sections revealed a very cellular tissue containing large thin-walled vessels. However, the cells appeared spindle-shaped, uniform in size and in staining quality of their nuclei. They showed a tendency to a vesiculated appearance and a kind of whorl formation. Occasional small calcified bony islets were seen. Around some of the bony islands a narrow rim of osteoid tissue was seen. Osteoblasts were noticed partly around the bony islets, partly in the connective tissue stroma proper.

The patient had an uneventful recovery and was discharged on the sixth postoperative day. He had two series of sinus films so far and there is no evidence of recurrence of the tumor one year after surgical removal.

In this case there was a cystically degenerated ossifying fibroma of the left antrum.

The clinical symptoms were an upward displacement of the left eye with obvious proptosis and displacement of the left lateral nasal wall against the nasal septum.

The outstanding feature of this case is the cystic appearance grossly and microscopically. Histologically it was a young-to-intermediate type of ossifying fibroma. Two cases of cystically degenerated ossifying fibroma of the frontal sinus have been reported. In 1902, Krogius[24] reported a case similar to this of the right frontal sinus under the heading "Spindelzellensarkom."

The case described by Benjamins,[11] in 1938, was a cystic osteoid fibroma of the left frontal sinus. According to Goegl[8] the cystic degeneration is probably due to circulatory disturbances and destruction of tissue.

CASE 4. O. M., an 18 year old colored male, was seen in the clinic, Research and Educational Hospital, on Dec. 10, 1953, with a swelling of the forehead. The patient stated that he was struck on the forehead with an iron bar nine years before. Approximately four years ago, he noticed a pea-sized swelling on the left side of his forehead, which grew very gradually to its present size of an egg. He complained of dull frontal headache of different frequency and intensity.

Physical examination revealed a well-developed, well-nourished male with an evident mass on the forehead (Fig. 4). This mass measured about one-half by two inches in diameter and was hard in consistency, painless and situated to the left of the midline in the supraorbital region.

Transillumination of the sinuses showed the left frontal sinus to be only partially clear.

X-ray of the skull revealed a silver-dollar-sized expanding lesion involving the area of the left frontal sinus and expanding both inner and outer tables in the diploic; however, the inferior and lateral part of the left frontal sinus was clear (Fig. 5). Complete blood study and urinalysis were normal.

On Feb. 1, 1954, an incision was performed through the left eyebrow, extending over the root of the nose and through the other eyebrow. A circular mass was seen in the left supraorbital region. The anterior wall was composed of very thin bone which could be readily depressed showing a cystic tumor containing serous fluid and lined with a thickened membrane. The tumor had eroded the bone down to the dura and caused a bulging into both frontal sinuses; but the sinuses were clear. The superior and lateral walls of the tumor consisted of soft spongy bone which was removed completely.

Microscopically, a cellular and very vascular connective tissue stroma was noticed. Extensive blood extravasations were seen. In the stroma, numerous calcified bony islets were found. Some of these bony islands were surrounded by osteoid tissue, lined by osteoblasts. Other areas revealed partly very cellular tissue, partly numerous small round amorphous calcified bone spicules fragmentally acellular. Between these spicules there were numerous mesenchymal cells, which showed various transitions between simple fibroblasts and osteoblasts.

Recovery was uneventful and the patient was discharged on the seventh postoperative day. There has been no recurrence ten months after removal.

A B

Fig. 4.

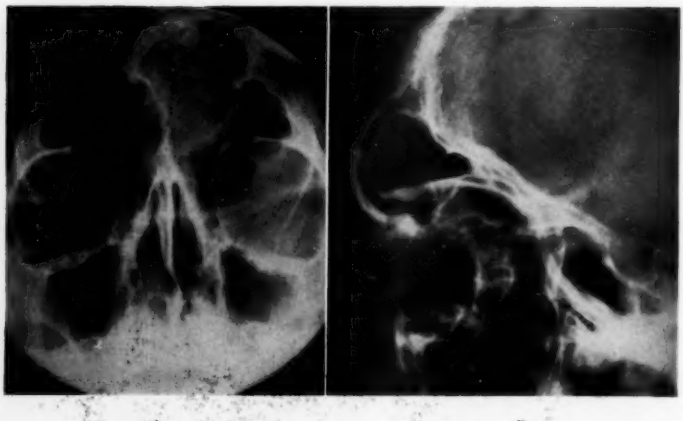
This was a case of cystic ossifying fibroma of the left supraorbital region involving the walls of the frontal sinuses. Two cases of ossifying fibroma similar to this one, but involving more the anterior portion of the frontal bone and the left frontal sinus have been separately described in 1951 by Ball[13] and Corbett.[14]

Histologically, this was an intermediate type of ossifying fibroma according to the classification of Smith and Zavaleta;[5] the dense fibrous stroma, the presence of islands of bone surrounded by osteoid tissue with prominent peripheral osteoblast layers account for this group.

SUMMARY

Four cases of ossifying fibroma involving the paranasal sinuses are described in detail.

The first is an ossifying fibroma of the maxillary sinus; the second is an ossifying fibroma of the maxilla involving the antrum; the third a cystically degenerated ossifying fibroma of the antrum; the fourth a cystic ossifying fibroma of the supraorbital region involving the walls of the frontal sinuses.



A B

Fig. 5.

The diagnosis presented both a clinical and a radiological problem; the surgical treatment was complete excision. Correlating the data from cases previously described and from our own experience conclusions are drawn concerning the clinical behavior, the radiological appearance, the histologic picture and the surgical treatment of these tumors.

CONCLUSIONS

1. Clinically, due to the dimensions of the sinuses and their slow growth, ossifying fibromas have a rather insidious development. As a rule these tumors are unsuspected until expansion of the bony walls of the sinuses calls attention to their presence. However, cases of persistent or frequently recurrent "sinusitis" which fail to respond to the usual treatment must receive a thorough evaluation and radiological study. The first sign of disease is generally a local swelling associated with local tenderness; displacement of the eye and functional disturbances may be present. In our series no lesions were noted other than in the cranial or facial bones, although extensive skeletal surveys were done. Similarly, no cutaneous, endocrine and biochemical alterations could be evidenced.

Ossifying fibromas are solitary lesions that do not metastasize and when completely removed do not recur. However, by virtue of extensive growth and pressure on surrounding vital structures they can be clinically malignant, though histologically benign.

2. Radiologically, ossifying fibromas are evident early as small, round swellings involving a cranial or facial bone and eventually projecting into the sinuses.

However, the non-specificity of the roentgenogram is demonstrated by the several differential diagnoses often offered by the radiologist. In particular, carcinoma, sarcoma, osteoma, osteochondroma, adamantinoma, Ewing's tumor, eosinophilic granuloma of bone, juvenile fibroma, osteitis fibrosa cystica, bone cyst, mucocele and osteomyelitis must be differentiated.

Massive fibromas may be present as benign expanding tumors with a picture of pressure atrophy of a part or whole of the sinus walls. In some cases, however, pronounced pressure atrophy can cause lesions resembling destruction, and the differentiation from a malignancy may be impossible on radiological grounds alone. In these cases the importance of a good biopsy in the diagnosis of these tumors cannot be overstressed.

3. Surgically, excision and curetment of the involved bones is the only therapy to be employed in these cases. X-ray therapy has been proved of very limited value. It is our feeling that in view of the benign nature of the lesion, massive resection and sacrifice of facial bones, with its accompanying disfigurement and high mortality, are not justified. Ossifying fibromas must be removed as thoroughly as possible surgically but without too great destruction of facial bones. No recurrence was evidenced in our cases with this treatment.

However, this does not necessarily mean that the patient should be considered cured. If the condition recurs in the follow-up, further partial or total resection is indicated.

4. Histologically, the uniformity of the nuclei and lack of mytosis accounts for the benign nature. The variability in the amount of fibrous and osseous tissue, the different character of the fibrous stroma and fibrous cells, the presence of osteoid tissue and of islands or trabeculae of bone, the degree of layering of osteoblasts and osteoclasts around bone or the presence of giant cells scattered throughout the stroma, are not only expressions of the continuous transformations these tumors undergo during their growth, but probably reflect an advancing histologic maturity parallel with the gradual increase in age of the patient.

However, these progressive histologic changes from a young to an intermediate and then mature type of ossifying fibroma into osteoma could be surely demonstrated only by a longitudinal study of

cases with repeated biopsies of facial or cranial bone lesion without removal of the tumor.

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LXVII

A NEW TECHNIQUE OF VESTIBULAR ROTATORY STIMULATION AND OF ELECTRICAL RECORDING OF NYSTAGMUS IN MAN

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The most adequate stimulation of the vestibular receptors, though chiefly of the semicircular canals, is obtained by rotation. Rotatory stimulation has often been disregarded for clinical investigations since it is not so easy to handle as is thermic stimulation, which allows the vestibular system to be stimulated separately on each side.

Among the various methods for rotatory stimulation of the semicircular canals, the best known is that of Bárány. This consists primarily of a sudden stop of rotation, allowing the observation of so-called postrotatory nystagmus of the eyeballs, which occurs as a result of a massive bending of the crista ampullaris. The original method of stimulation of Bárány appears today inadequate in many regards (intensity, speed) for physiological and clinical purposes. Therefore we needed a more adequate method for rotatory stimulation of the semicircular canals, which would allow us to observe successively the effects of an initial acceleration (Beschleunigung or positive acceleration) and then the effects of deceleration, that is of a progressive slowing of the rotation (Verzögerung or negative acceleration).

These new principles of stimulation imply the use of a rotating chair activated by a motor allowing a rotation at progressive regular speed (constant acceleration during the period of acceleration) and a rotation at regular regressive speed during the period of deceleration.

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On the other hand it is no longer adequate to observe directly the vestibular nystagmic reaction of the eyeballs only during the post-rotatory period. An exact analysis of vestibular nystagmic reaction is possible by means of an electrical recording. Myers,[29] Jung[19] and Jung and Tönnies[20] have already shown the principles and the clinical applications of an electrical bitemporal recording of the nystagmus. Monnier and Hufschmidt[34] emphasized later the fact that an adequate analysis of the nystagmus is only possible if the nystagmic reaction is recorded from each eye simultaneously and separately. They studied the best positions of the electrodes and of the leads in the neighborhood of the eye for this purpose. Since the distribution of the corneo-retinal electrical field around the orbit changes according to each movement of the eyeball it is possible to detect by means of highly sensitive amplifiers and oscillographs the smallest ocular jerks. The advantage of this method lies in the fact that it provides more accurate and extensive information than the mere observation of the eyes. The nystagmus can even be recorded in the dark, thus without any optic afference. The same electronic technique has already been applied successfully to the analysis of the vestibular nystagmus in animals by Montandon and Monnier,[37] Montandon, Monnier, Croci and Brunner[38] and Monnier and Laue.[35]

METHOD

a) *Technique of liminal rotatory stimulation of the vestibular receptors.* Steinhausen[44] and later Dohlman[6] demonstrated that a slight rotation in the horizontal plane induces a progressive bending of the cupula ampullaris of the horizontal canal in the direction of the endolymphatic stream and that a sudden stop brings the cupula rapidly back to its resting position. If, instead of a slight and brief stimulation, the rotation lasts longer at a constant speed, the cupula ampullaris returns to its resting position, owing to its elasticity. Later, stopping the rotation induces a new bending of the cupula in the opposite direction followed by a series of aperiodic deflections during a long period. Since the deflection of the cupula is responsible for the nystagmic reaction and since this deflection is a consequence of the acceleration or deceleration of the rotation, it seems obvious that we will be able to observe two distinct periods of the nystagmus in opposite directions: An acceleratory nystagmus and a deceleratory nystagmus. Our purpose was to apply an acceleration and a deceleration of minimal intensity in order to determine the threshold of the acceleratory and deceleratory nystagmus. The intensity of the rotatory stimulus is defined by the acceleration of the movement of rotation, as given by the formula:

$a = \frac{v}{t} = \frac{n^\circ}{t^2}$, or in seconds $\frac{n^\circ}{sec^2}$; where a = acceleration; v = velocity; t = time and n = number of angular degrees.

This new method of vestibular rotatory stimulation has been called by us "the method of liminal rotatory stimulation" (LRS). It involves a succession of clockwise and anticlockwise rotations at progressively increasing and decreasing speeds, separated by a period of rotation at a constant speed. This constant speed represents the maximal speed of the rotation and amounts usually to 90°/sec., that is one rotation within four seconds. This constant speed was applied for the rather long period of three minutes. The acceleration and the deceleration have the same magnitude during the same rotation. In order to measure the threshold, the intensity was increased successively from 0.4°/sec² to 7°/sec².

The liminal rotatory stimulation was applied in our experiments by a special electrical motor with a hydraulic transmission system and a servomotor allowing an automatic control of the acceleration and deceleration, the intensity being regulated by means of a scale which gives the degree of acceleration at once. Acceleration and deceleration were kept strictly constant during the whole experiment.

b) *Electronystagmography* (*ENG*). Previous experiments of electrical recording of nystagmus in man show that the best results are obtained with a horizontal lead along the inferior edge of the orbit for detecting the horizontal nystagmic jerks, and a vertical lead between the superior and inferior edges of the orbit for detecting vertical jerks (Monnier and Hufschmidt,[34] Monnier,[33] Dieterle and Berger[5]). The axis of the vertical lead must run three to four millimeters medially to a vertical axis passing through the center of the pupil (Fig. 1). The optimal distance between the supraorbital and infraorbital electrodes must not exceed six centimeters and the distance between the two infraorbital horizontal electrodes must not exceed two centimeters. Since the distance between the two electrodes of the vertical leads is two to three times greater than that of the horizontal lead, it is necessary to reduce the magnification (attenuation amplification) for the signals detected in the vertical lead. The polarity in which the electrodes are connected to the entrance of the amplifying channel has a great practical importance. We recommend the adoption of a convention according to which abduction of the eyeball (temporalwards) gives rise to an upward electronegative deflection on the electronystagmogram (ENG). An adduction or

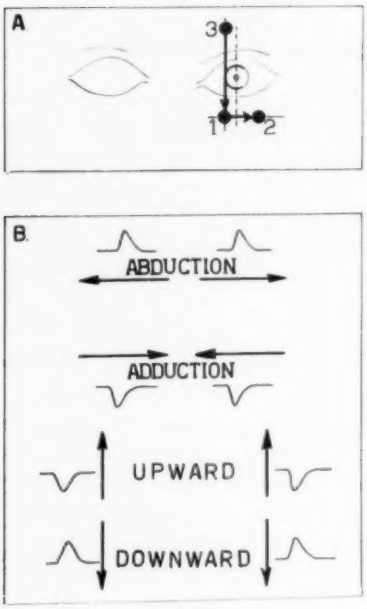

Fig. 1.—A) diagram of an adequate placement of the electrodes for electronystagmography. B) Code for the interpretation of the electrical deflections. Above, corresponding horizontal nystagmic jerks. Below, corresponding vertical nystagmic jerks.

convergence movement (nasalward) on the contrary is then expressed by a downward electropositive deflection. Among the vertical movements the upward gaze is expressed by a downward electropositive deflection; this occurs in every blinking reflex with its synergic shifting of the eyeballs upward (Bell phenomenon). On the other hand an upward electronegative deflection corresponds to a downward deviation of the gaze. The schema (Fig. 1) shows that, if standard electroencephalographic instruments are used for electronystagmography (Grass, Model III, Reega, Edison and Swan, Schwarber etc.) the convention is correct when the electrodes of the horizontal lead are connected according to the polarity 1 - 2 and the electrodes of the vertical lead according to the polarity 3 - 1. On some other EEG instruments, the older Offner type A, for instance, the polarity was just reversed.

Every experiment of electronystagmography starts with a calibration test. The patient sits comfortably on a chair fixed with the amplifiers on the rotation plate in the screened cage. He gazes at the center of a perimeter placed in front of his face. He is now asked to look laterally (temporally) at a luminous point located 2.5° from the initial fixation point. This lateral movement of the eyeball is recorded electrically. The eye turns back to the initial point and the patient is now asked to gaze successively at 5°, 10°, 30° and 40°, while the eye returns each time to the initial point. If the technique used corresponds to the rules previously quoted and if the instruments have amplification and discrimination power corresponding to the recommendation of the International Federation of EEG it is possible to detect minimal movements of the eyeball with an angular deviation inferior to 2.5°. In the deflections recorded with an optimal time constant, the quick component of a nystagmic jerk is expressed by the steep phase of the deflection and the slow component by the slow return of the deflection to the isoelectric line. An exact analysis of this slow "vestibular component" of the nystagmic jerk is possible if the electronystagmogram is recorded with amplifiers at a time constant above 0.3 sec.

It was found more convenient to have the motor driving the rotating plate outside of the screened and sound-proof cage to avoid microphonic and electrical artifacts as well as the disturbing effect of noise on the patient. Furthermore, the best results were obtained when the screened cage was kept in the dark.

RESULTS

We intend to analyze first the features of the nystagmic reaction to rotatory stimuli of very small intensity (1 degree/sec^2 or less), and then the alterations of the nystagmic reaction according to rotatory stimuli of increasing intensity. It will be shown that the nystagmic reaction which occurs during the acceleratory and deceleratory stimulation may last during the period of rotation at constant speed (postacceleratory nystagmus) and during the period which follows the end of the deceleration (postdeceleratory nystagmus, previously called postrotatory nystagmus by Barany and other investigators). Finally we will keep in mind that some symptomatic alterations may be observed after the postacceleratory nystagmus during the period of rotation at constant speed.

a) *Nystagmic reaction to rotatory stimulations of minimal intensity* (Fig. 2). If we apply a perrotatory stimulation with a constant acceleration of 1 degree/sec^2 in anticlockwise direction we observe after a period of eight seconds at least the first regular deflec-

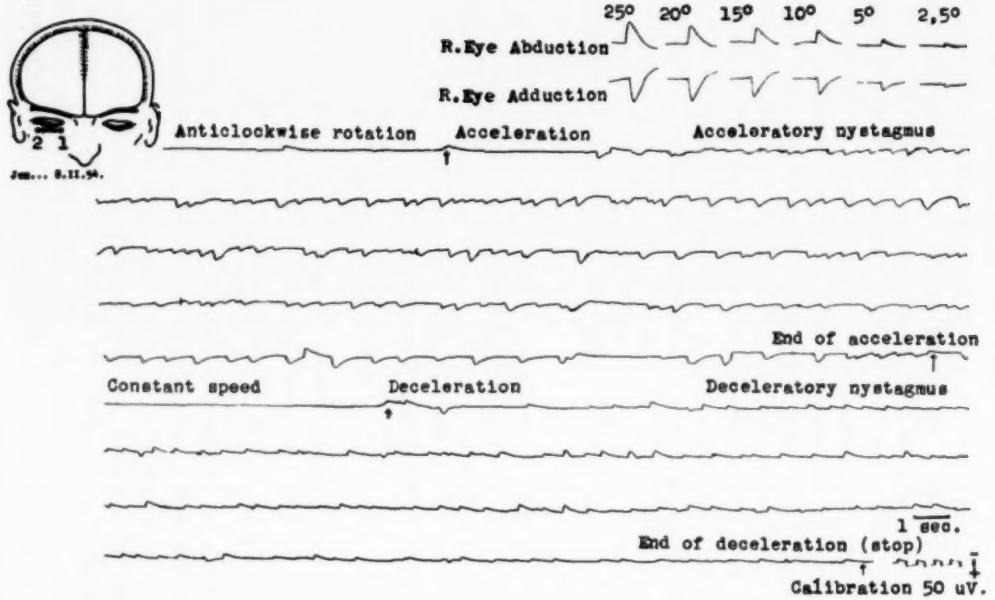

Fig. 2.—Perrotatory nystagmic response to an anticlockwise weak rotatory acceleration (perrotatory acceleratory nystagmus) followed by the nystagmic response to weak rotatory deceleration (perrotatory deceleratory nystagmus). Disappearance of the nystagmus during rotation at constant speed and toward the end of deceleration. Constant acceleration: 0.8°/sec^2. Rotation at constant speed 90°/sec. Constant deceleration 1°/sec^2 (weak stimulus).

tions of the acceleratory nystagmus. In response to an anticlockwise rotation, we obtain from the right eye a nystagmic reaction characterized by a quick phase directed to the left, that is by rapid adductory movements expressed by a downward deflection of the oscillogram (electropositive potential). The nystagmic reaction during this acceleratory stimulation consists of a succession of deflections with a rapid downward phase and a slower return to the isoelectric line. The former represents the quick phase and the latter the slow phase of the nystagmus. The record shows clearly that the amplitude and frequency of the nystagmic jerks vary during the acceleratory period. We may measure a series of deflections at higher frequency with lower amplitude (frequency 2-3/sec., amplitude 50 microvolts) or a series of deflections at lower frequency (1/sec.) with higher amplitude (100 u.V. or more). This acceleratory nystagmus ends about 100 seconds after the beginning of the acceleration. It is followed by a period during which there are only sporadic, nonperiodic positive deflections of smaller significance, which correspond to the end of the acceleratory excitation of the cupula. Since the speed of the rotation at constant speed was always 90°/sec. and since the acceleration lasted about 110 seconds we may conclude that the stimulation was produced in this experiment by a constant acceleration of 90°-110°/sec or 0.8°/sec^2, which represents the threshold of the acceleratory nystagmus, as shown later on.

After the end of the acceleratory stimulation no deflection can be detected, though the rotation continues at a maximum constant speed. The receptors of the cupula are no longer stimulated since the horizontal semicircular canals are rotated at the same speed as the endolymph in which no current occurs. This period of rotation at maximal constant speed without stimulatory effect and without electrical deflections lasts about three minutes. We will see later in cases of stimulation with increasing intensities that the acceleratory nystagmus may continue during this period of rotation at constant speed. We propose to call this reaction "postacceleratory nystagmus."

Now if we brake the speed of the rotation progressively, inducing a deceleration from 90°/sec. to 0, we observe again, after a so-called latency of about five seconds or less, a new series of upward deflections (electronegative potentials) corresponding to rapid abductory jerks of the right eye (quick phase of the deceleratory nystagmus). These deflections show a somewhat smaller amplitude than those of the acceleratory nystagmus (90 to 80 u.V.). The frequency of the deflections varies between one and two per second. Since this deceleratory nystagmus lasts about 84 seconds or more and ends at about the same time as the stop of deceleration, we may consider that the

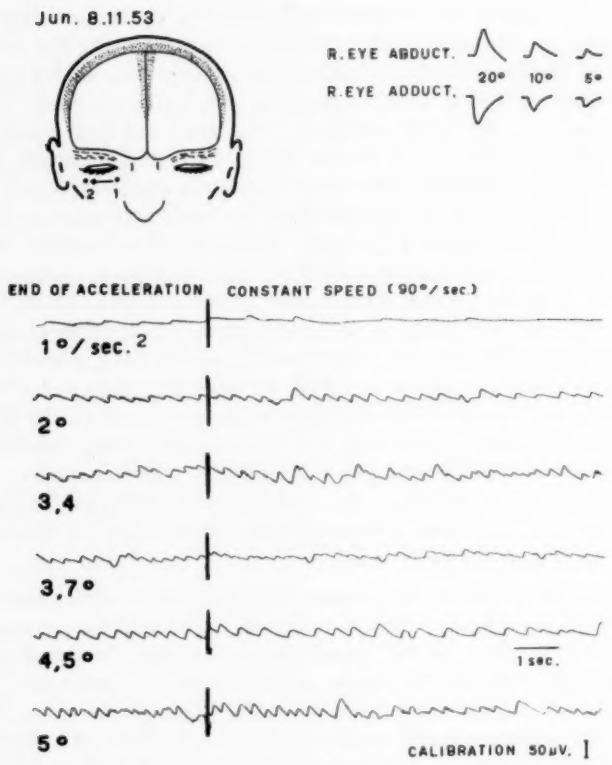

Fig. 3.—Perrotatory nystagmus during and after acceleration at different degrees (clockwise rotation). Nystagmus during clockwise rotatory stimulations of increasing intensities (1°/sec.² ----- 5°/sec²). Changes occurring in the nystagmus during the acceleration and at the end of acceleration, when the speed of the rotation becomes constant (90°/sec). Optimum of the amplitude of the acceleratory nystagmus at stimulation intensities at 2°-3,4°/sec²). The overlapping of the acceleratory nystagmus on the period of rotation at constant speed is much poorer and shorter than after stimulation at strong intensities. Lower rate of the nystagmic frequency after the end of acceleration (4,5°/sec²) during the rotation at constant speed.

threshold of the deceleratory nystagmus amounts to 90°-84°/sec : about 1°/sec².

b) *Nystagmic reaction to rotatory stimuli of increasing intensity.* (1.) *At different degrees of acceleration* (Fig. 3). If the vestibular system is stimulated in the same subject on the same side by means of rotation at different degrees of acceleration (increasing intensity), at 1°/sec², 2°/sec², 3.4°/sec², 3.7°/sec², 4.5°/sec², 5°/sec², the ENG recorded during the 25 last seconds of acceleration shows the following features:

At very low intensity (1°/sec²) the nystagmic reaction shows a low frequency (1-1.5 cycle/sec) and a low amplitude (20 u.V. or less), with progressive extinction toward the end of acceleration. After the end of acceleration, during the rotation at constant speed, some very weak nystagmic jerks may be observed during the first five seconds.

During acceleration at 2°/sec² or 3.4°/sec² the ENG is characterized on the contrary by rapid and high deflections at a rate of 3 to 3.5 cycles/sec (25-45 u.V.), representing the optimal conditions for electronystagmography. After the acceleration the nystagmus lasts for 20 seconds at least with a decrease in frequency (2.5 to 2 cycles/sec) and amplitude (10 u.V.).

At higher intensity (4.5°/sec², 5°/sec²) the record becomes irregular since two different components may alternate or interfere: A component of low amplitude (15 u.V.) and high frequency (4 cycles/sec) and another component of higher amplitude (40 u.V.) and low frequency (2 cycles/sec). Furthermore the record is disturbed by isolated high random deflections apparently due to aperiodic movements of the eyeball. After the end of the acceleration, during the rotation at constant speed the slow rhythms seem to become predominant. In other words deflections of higher amplitude, smooth rise (slow component of the nystagmus) and long duration, are more numerous. This postacceleratory nystagmus again lasts about 20 seconds. It seems that the nystagmus is more irregular and more polyrhythmic when stimuli of higher intensities are used (acceleration 4.5°/sec² and 5°/sec²) and that the optimal condition is a stimulation at 2°-3,4°/sec² acceleration. The duration of postacceleratory nystagmus during rotation at constant speed amounts to 20 seconds as a maximum.

Nystagmic reaction to rotatory stimuli of increasing intensity (2.) *At different degrees of deceleration* (Fig. 4). Let us compare the nystagmic reaction toward the end of a weak deceleration (0.7°/

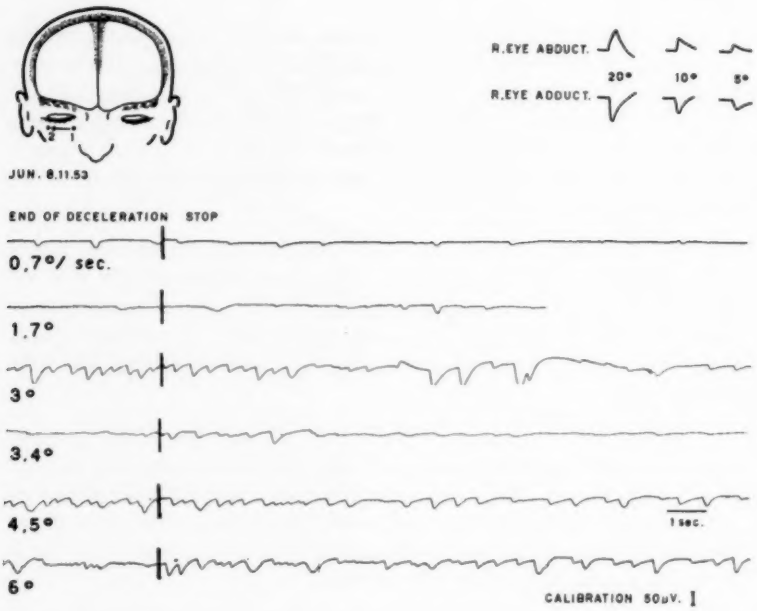

Fig. 4.—Perrotatory nystagmus during deceleration at different stimulation intensities (0.7°/sec² to 6°/sec²) and postrotatory nystagmus. Clockwise rotation. Towards and after the end of deceleration overlapping of the nystagmus with an optimum at intensity 3°/sec² (post-deceleratory and post-rotatory nystagmus), similar to the post-acceleratory nystagmus in Figure 3.

sec²) with the reaction after moderate deceleration (3°/sec²) or after a deceleration at strong intensity, 4.5° and 6.5°/sec². Stimulation at 3° provides the best record with deflections of high amplitude (40 u.V.), moderate frequency (3 cycles/sec) followed by a rather non-rhythmic behavior. After the end of the deceleration the postdeceleratory nystagmus (previously called "postrotatory nystagmus") lasts 15-20 sec. sometimes with irregularities of the base-line due to shifting of the gaze. According to convention the downward direction of the deflections indicate that the fast component of the nystagmus in the right eye moves to the left (nasalwards).

If a weak deceleration stimulus is used (0.7°/sec², first record) the nystagmus toward the end of deceleration has a low frequency, about 1 cycle/sec. or less and a low amplitude (25 u.V.); after the stop the nystagmus keeps the same characteristics, with a progressive slowing until it disappears after 13 sec.

At strong intensities we observe the same irregular polyrhythmic behaviors of the deceleratory nystagmus with a mixture of fast and slow rhythms. After the end of the deceleration, the postdeceleratory nystagmus presents the same tendency to a predominance of slow rhythms, with high deflections and a duration of 18 sec.

We may conclude from the comparison of these various nystagmic responses to stimuli of different intensities that the qualitative and quantitative features of the acceleratory and deceleratory nystagmus are very similar since the physical conditions of stimulation with our technique are equal. However Figures 2 and 3 show that acceleratory nystagmus is more regular than deceleratory nystagmus; this is due to the fact that the subject is less tired during the acceleration period than the deceleration period. Finally it must be stated that the optimal conditions for a quantitative analysis of nystagmus are found during the middle of the acceleration or deceleration and toward the end of the acceleratory nystagmus when acceleration at moderate intensity (3 to 3.5°/sec^2) is being applied.

POSTROTATORY NYSTAGMUS AS A SPECIAL CASE OF DECELERATORY NYSTAGMUS

It has been stated previously that nystagmus may last a certain time after the end of rotatory stimulation. In the case of deceleration nystagmus, the activity occurring after the end of rotation corresponds to the postrotatory nystagmus described by Bárány. Indeed a sudden stop—that is a stop within one second—during a rotatory stimulation at a rate of 10 rotations within 20 seconds is equal to a deceleration at a rate of 180/sec^2, in a short time.

Figure 5 illustrates the behavior of the eyeballs toward the end of a clockwise rotation at constant speed of 90°/sec. (5 turns within 20 seconds) with its complete lack of nystagmic rotation and a sudden stop of the rotation. One second after the stop the nystagmic jerks occur, expressed by a regular succession of downward deflections, corresponding to adduction movements of the right eye (quick component of nystagmus reaction toward the left). These deflections have an average amplitude of six millimeters corresponding to a voltage of 50 u.V. This postrotatory nystagmus lasts for 48 seconds with decreasing amplitude and frequency. In fact, the frequency of three deflections per second decreases to one deflection per sec. or less, toward the end of the stimulation. The reaction then loses its regularity and the record becomes polymorphic.

COMMENT

A new technique of vestibular rotatory stimulation and of electrical recording of the nystagmus in man, previously described, allows

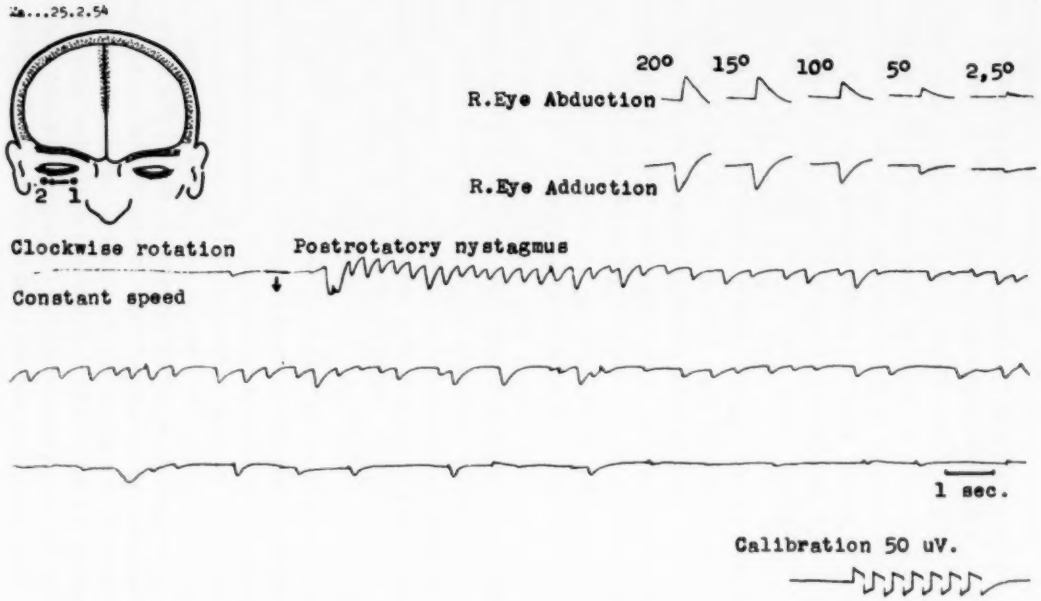

Fig. 5.—Classical post-rotatory nystagmus following sudden stop after clockwise rotation at constant speed 90°/sec. Deflections of high amplitude (10° = about 50 u.V.). Acceleration: 1°/sec^2. Constant speed 90°/sec. Sudden stop 90°/sec (strong stimulus).

an exact analysis of the excitability of the cupula ampullaris during perrotatory stimulations (acceleratory nystagmus, during the period of rotation at constant speed, deceleratory nystagmus) as well as during postrotatory stimulation. The responses may be exactly defined in terms of amplitude of the nystagmic jerks, voltage of the electrical deflections, frequency, overlapping of the nystagmus during the postacceleratory and postdeceleratory periods and duration of the postrotatory nystagmus following a sudden stop of the rotation.

The great advantage of our technique over the previous methods of vestibular stimulation (Bárány[2]) lies in the fact that it allows us to measure *the threshold of vestibular excitability*. The technique is called therefore "a technique for liminal stimulation of the vestibular receptors." The threshold of the vestibular system had been previously measured subjectively on the basis of the sensation of rotation which appears during a very weak and slow rotation of human subjects. The threshold values determined with subjective methods are not considered in this paper. We may quote, however, Mach[25] who found the first rotatory sensation at a threshold of $2°/sec^2$, more recently Buys,[3] Arslan[1] at a lower value, finally Hilding[16] at $0.5°/sec^2$.

It was important to develop a method which allows an objective measurement of the vestibular perrotatory excitability. For this purpose we found that the best procedure consists of six successive measurements: a) Determination of the threshold of the nystagmic response to weak acceleratory stimuli of progressive intensity such as $1°/sec^2$, $3°/sec^2$ and $5°/sec^2$; b) determination of the threshold of the nystagmic response to weak deceleratory stimuli of progressive intensity such as $1°/sec^2$, $3°/sec^2$ and $5°/sec^2$.

The average value of the vestibular threshold obtained with our technique of liminal stimulation amounts to $0.8°/sec^2$. Below $0.5°/sec^2$ no nystagmic response was observed. Stronger stimuli have been disregarded as unphysiological since they alter the excitability of the receptors for a long time and make the repetition of stimulations unreliable.

Furthermore we found it useful to add as a standard test a weak acceleratory stimulation of $1°/sec^2$ followed by a three-minute period of rotation at a constant speed and finally by a sudden stop inducing a postrotatory nystagmus.

SUMMARY

1. A method of liminal stimulation of the vestibular receptors has been worked out allowing the measurement of the threshold of

the ocular nystagmus in man. The nystagmic reaction elicited by a sudden interruption of the rotation at high intensity (postrotatory nystagmus) is a massive reaction to rather unphysiological stimulation. Therefore attention has been paid chiefly to the responses following an acceleratory and a deceleratory rotatory stimulation of weak intensity ($1°/sec^2$, $3°/sec^2$, $5°/sec^2$).

2. The stimulating technique consists of a rotating disk on which the patient is sitting. This disk is moved by means of an electrical motor with servomotor system and hydraulic transmission, allowing rotation with progressive speed, constant acceleration and deceleration at different intensities, clockwise or counterclockwise.

3. The nystagmus is recorded electrically according to the technique of Monnier and Hufschmidt. Electronystagmography = ENG. The electrical potentials corresponding to the deflections of the eyeball are derived from horizontal or vertical orbital leads, the patient sitting in the dark, in a silent and shielded room. The recording instrument (electroencephalograph Reega, Paris) is placed on the rotating disk behind the patient.

4. The method has allowed us to determine objectively the average threshold of the ocular nystagmus to an acceleratory and deceleratory rotatory stimulation of the vestibular receptors (cupula ampullaris). The average value amounts to: $0.8°/sec^2$. Below $0.5°/sec^2$ no nystagmic response whatever was observed.

5. Stimulation with weak acceleratory rotatory stimulus of $1°/sec^2$ followed by a three minute period of rotation at constant speed and a sudden stop was used as a standard test. This permitted an analysis of the perrotatory acceleratory nystagmus at the same time.

6. The most important advantage of this method is the application to clinical investigations of the recent physiological acquisitions concerning the semicircular canals. It allows by means of electronystagmic records the comparison of the normal and pathological thresholds of the nystagmic reactions in all disorders of the vestibular functions.

We are very much indebted to Professor Max Linder of the Institute of Statistical Mathematics, for the accurate calculation of the average values.

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LXVIII

CHONDROSARCOMA OF THE NASAL CAVITY

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Chondrosarcoma, as the name indicates, is a malignant tumor of connective tissue origin arising from cartilage. In 1952, Lawson[1] reported the clinical and pathological findings of a 76 year old woman treated by surgery and x-ray therapy for a chondrosarcoma apparently arising in the nasal septum, filling the right side of the nose and extending into the right maxillary sinus. Ten years earlier, a chondroma had been removed from the left side of the nose.

Interestingly, at the time of his report, we had under our care and observation a woman who also had a long history of nasal obstruction produced by a tumor mass of cartilaginous origin. There had been a continuous, progressive growth of the mass with development of symptoms and findings reflecting the pressure and destruction produced by it, as it extended locally beyond the confines of the nasal cavity to invade the cranial cavity.

Since these tumors are rare in the nasal cavity, we feel that a report of the clinical findings correlated with the x-ray changes, histopathological findings and gross pathology would be valuable.

In 1938, Ringertz[2] presented a review of the pathological findings in 391 cases of tumors, chiefly malignant, of the nasal and paranasal cavities and maxilla. This large series did not include a single case that he was able to diagnose as chondrosarcoma and there were only 2 cases which he felt could be classified as enchondroma. Further, he reviewed reports of 1101 additional cases in the literature and found only 5 cases of chondrosarcoma. His review revealed that nasal and paranasal tumors made up chiefly of cartilaginous tissue are rare—only 60 to 70 such cases reported altogether—and most of these were enchondromas.

The diagnosis and classification of tumors of cartilaginous structure often present many difficulties. These growths belong to the

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large group of tumors which are classified as of osteogenous origin. They arise from tissues which participate in the formation of normal bone. During the course of development certain bones form directly from mesenchymal tissue without an embryonal cartilaginous stage. Most of the bones of the body, however, are preceded by a temporary cartilaginous model, later replaced by bone tissue.[3]

In the skull, the sphenoid and ethmoid bones and nasal capsule are among those which pass through a cartilage phase. Tumors made up of cartilage may originate in persistent cartilage rests remaining within these bones after ossification has occurred. Most of the cases of enchondromas reported to develop in the head region occur in the ethmoid bone and adjacent nasal region. The majority of cartilaginous tumors, benign or malignant, develop in the long bones, hands, feet, ribs and sternum, or pelvis. Geschickter and Copeland[4] state that tumors in this group include osteochondromas, chondromas or enchondromas and chondrosarcoma or chondromyxosarcomas.

Osteochondromas are benign tumors often appearing as exostoses and having a thin cartilaginous cap covering the bulk of the mass made up of cancellous bone. They usually arise from the end of the long bones and may attain a large size.

Chondromas or enchondromas are classified as benign cartilaginous tumors generally reported to consist of cartilage of relatively adult type. They are of central origin in the bone usually occurring in the bones of the hands or feet, ribs, sternum, spine or pelvis. In the skull, they most often occur in the ethmoid bone but may develop in the sphenoid, nasal septum, or lateral nasal wall. Boyd[5] reports that grossly they are usually lobulated, often having a gelatinous character and showing cystic changes in the larger tumors.

Microscopically they may be composed of fairly normal adult cartilage, with a matrix of hyaline material and showing cartilage cells arranged in pairs or tetrads. Ringertz,[2] however, states that the cartilage tissue of enchondromas has a characteristic histologic picture differing from normal cartilage by its atypical cells; the cells are usually larger than those of normal cartilage, and irregularly arranged, with large vacuoles about their nuclei and showing occasional mitoses. The intercellular substance is poor in fibers and variable in amount. Some of the cells may be stellate and scattered in the hyaline intercellular substance rather than collected in groups.

Though these tumors may show a benign histologic picture, they apparently have a great growth capacity, destroying bone about them by pressure erosion. In the nasal region such tumors grow slowly

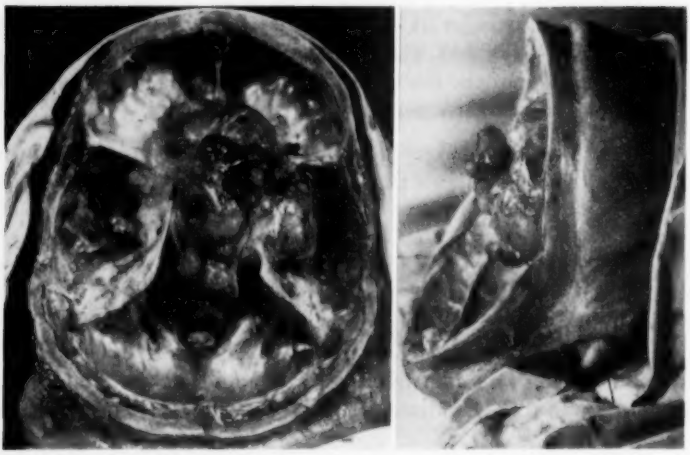

Fig. 1.—A. Post mortem view of interior of the base of the skull revealing the massive invasion by the tumor into the anterior and middle fossae to the foramen magnum. B. Lateral view showing the height of the intracranial tumor mass above the base of the skull. Note the smooth intact margins and lobulated gross appearance.

and expansively, usually recurring after incomplete removal. X-ray examination usually reveals evidence of bone destruction and may show a mass involving completely the nasal or paranasal cavities.

Chondrosarcoma shows certain histologic as well as clinical features which help differentiate it from chondroma. These tumors are often classified as primary or secondary. The primary type is the one which arises from relatively undifferentiated cells of the perichondrium. The secondary type, which is the more common, often arises from cartilage rests remaining in the central portion of the bone after ossification has occurred. Coley[6] states that secondary chondrosarcoma is nearly always seen in adults; it results from a malignant alteration in the cells of a cartilaginous exostosis, a central chondroma or rarely in a patient with multiple skeletal defects. Lichtenstein and Jaffe[7] found that chondrosarcoma differs from osteogenic sarcoma in that the former almost always develops from full-fledged cartilage while the latter develops from more primitive tissue such as bone-forming mesenchyme.

In addition, they report that chondrosarcoma never shows neoplastic osteoid tissue and bone evolving directly from a sarcomatous

stroma. Because there are definite microscopic differences and very distinct patterns in clinical behavior between these two groups of osteogenous tumors, the Registry of Bone Sarcoma of the American College of Surgeons, in 1939, recognized chondrosarcoma as a distinct tumor group.

Ewing[8] states that while in general the more cellular and softer tumors are the more malignant, both clinical and histologic features should be regarded in applying the term chondrosarcoma. The structure of invasive chondromas does not always suggest malignancy.

Just as in the chondromas, there is often a marked variation in the growth rate and degree of malignancy of the chondrosarcomas. Their histologic appearance is also quite variable. Various portions of the same tumor mass may show different histologic pictures ranging from areas with much fibrous or myxomatous material, through mature cartilage to regions with irregular calcification. At the periphery there may be more cellular and anaplastic areas.

Though many reports suggest that it may be difficult to distinguish benign from malignant cartilaginous tumors on a histologic basis alone, Lichtenstein and Jaffe[7] state that there are "subtle but tell-tale histologic indications of malignity already present even in the early stage of the evolution of a chondrosarcoma." The features suggesting malignancy include: 1) Many cells with plump nuclei, 2) more than an occasional cell with two such nuclei, and 3) giant cartilage cells with large single or multiple nuclei.

Clinical evidence of malignancy includes the progressive destructive course, the gross appearance of the tumor mass, the tendency to invade, and the development of metastases. Often chondrosarcomas remain only locally invasive for years. Primary chondrosarcomas arising from the perichondrium usually occur in young patients and a striking feature is the development of metastases and extension into the veins. The secondary chondrosarcomas usually develop in patients in an older age group, most often from 30 to 50 years of age.

The most common sites of occurrence are the long bones, the pelvis and ribs. X-ray examination usually reveals a destructive process in the bone involved. Though chondrosarcomas and osteogenic sarcomas show a different clinical picture, Pendergrass et al,[9] reviewing the x-ray findings in a series of these cases, found that the diagnosis of the pathologic type of malignant bone tumor cannot be made with certainty from the roentgen examination alone.

Clinically, chondrosarcomas may also vary greatly in their behavior. Wirth and Shimkin[10] reported a case of chondrosarcoma of

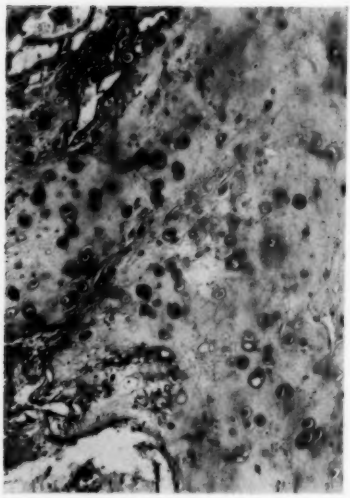

Fig. 2.—Photomicrograph showing the characteristic cartilaginous appearance of tumor tissue from early biopsy.

the nasopharynx occurring in a 16 year old boy, showing relatively rapid, progressive extension despite surgery, x-ray therapy, and radon seed implantation. There was intracranial extension of the tumor as well as pulmonary metastases. Warren[11] reported the findings of another case in a 32 year old patient whose primary developed in the pelvis with rapid progression despite deep x-ray therapy. Here there was direct extension into the veins with growth of the tumor intravascularly along the common iliac vein upward into the inferior vena cava and downward into the femoral vein. A tumor embolus which broke off passed through the right side of the heart, reached the pulmonary artery and developed further at this site.

In both of these cases response to irradiation therapy was poor. On the other hand, Lawson's patient showed some response to limited surgical therapy followed by deep x-ray therapy.[1] Phemister[12] also reported a case of chondrosarcoma of the humerus treated by local resection followed by 7500 r. of deep x-ray therapy given within 60 days with a five year follow-up showing the patient free of clinical or x-ray evidence of chondrosarcoma.

It is generally agreed, however, that the treatment of choice for chondrosarcoma is wide surgical excision when such treatment is

feasible. The diagnosis of such tumors occurring intranasally can usually be established by biopsy. Even those which appear histologically benign are usually clinically malignant because of their very location. Since they grow expansively within a limited cavity, both chondromas and chondrosarcomas progress by steadily eroding surrounding bone and destroying vital structures.

Early and wide surgical removal, though necessarily sacrificing surrounding tissue, may halt the progression of the tumor which has the potentialities of being so much more indiscriminatingly destructive. Bordely and Longmire[13] described one approach to the nasal cavity through a lateral rhinotomy. Priest and Kucera[14] have discussed in some detail the value of the Fergusson type of external approach to the nasal cavity and maxilla.

We have found that the Fergusson incision not only provides a good exposure of the area to be explored, but gives excellent cosmetic results which are admittedly, of course, a secondary consideration in the management of such malignancies.

REPORT OF A CASE

In April, 1945, Mrs. A. T., aged 40, was examined because of the complaints of nasal obstruction, fullness in the ears, repeated colds and recurrent cough. At that time a diagnosis of hyperplastic rhinitis was made and symptomatic therapy given.

Two years later, in May, 1947, she returned, complaining of severe headache which had occurred intermittently during the past year and a half, but which was much worse in the past few days. In addition, she had noted increasing nasal obstruction.

Examination revealed a large, pale, firm mass in the region of the left middle turbinate, with some edema of the left upper eyelid. The remainder of the ear, nose and throat examination showed no other significant abnormal findings.

Biopsy of the mass was attempted and some "gelatinous appearing material" was removed. X-rays of the paranasal sinuses showed a clouding of both ethmoid regions with clouding over the left antrum and membranous thickening of the right antrum.

The pathological report of the tissue removed from the nose was chondrosarcoma. Additional material was removed with a snare for further microscopic study, relieving the nasal obstruction and severe headache.

At this time examination of the eyes revealed normal findings in the cornea, sclerae and conjunctivae, with normal external ocular

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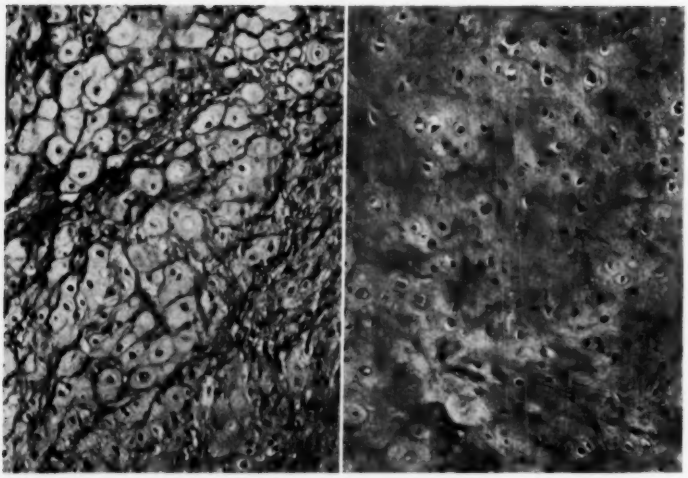

Fig. 3.—Photomicrographs of two areas of tumor tissue taken from the mass at post mortem examination showing less characteristic differentiation and varied structure.

movements. Vision was 20/20 on the right eye and 20/80 on the left. Fundi were normal. Pupils were normal, equal and reacted to light and accommodation.

Surgical treatment was recommended, but with the relief of the symptoms, this recommendation was not accepted. Repeated follow-up examinations during 1947 showed the nasal passages to be quite clear without evidence of tumor tissue.

Almost a year later, in October 1948, the patient returned again with marked nasal obstruction and examination revealed a firm mass filling the upper straits of the left side of the nose.

Laboratory studies showed RBC 4.79 million/cu mm, WBC 9,300, Hb 13.4 gms. Serology—negative. Urinalysis—normal findings and chest x-ray normal.

Biopsy of the mass in the nose was reported as chondroma. The patient was hospitalized, and, under local anesthesia, intranasal removal of the mass with exenteration of the ethmoid on the left was accomplished. The pathological diagnosis at this time was myxochondroma. However, on reviewing the slides, the pathologist added the following note:

"In spite of a relatively benign histologic picture it is difficult to predict the clinical behavior of these tumors. Many with a benign histologic picture prove to be clinically malignant."

Following this surgery the patient was again relieved of her symptoms and returned for follow-up examination only at irregular intervals.

In November, 1950, two years after the intranasal surgery, she suddenly developed a clouding of vision in the right eye, with intermittent headache in the right frontal region and gradually increasing nasal blockage. The fundi appeared normal and the intraocular tension was normal bilaterally. The visual acuity in the right eye had decreased from 20/20 to 20/80. Paranasal sinus x-rays were repeated, revealing clouding and an expanding lesion in both ethmoids and a portion of both antrums with some clouding in the region of the sphenoid sinus. The vision gradually improved and the patient was not seen again for a year.

In November, 1951, her symptoms became much worse with persistent headache, nasal obstruction and blurring vision on the right. A large mass was found involving both nasal passages. Surgical exploration was urged but she did not enter the hospital until several months later, in April, 1952, when an external approach to the antrum and nasal cavity was made through a Fergusson incision. The firm, gray, lobulated tumor mass was found to involve not only the right antrum and both sides of the nose but extended upward and backward between the orbits to involve the cribriform plate and sphenoid region, where it was clearly inoperable. Microscopic study of the tissue removed revealed chondrosarcoma.

The growth of mass continued slowly but steadily until the patient's death in August, 1954, almost nine years after her first complaint of nasal obstruction and seven years after biopsy was reported to show chondrosarcoma. There was progressive loss of vision, first in the right eye, then in the left, until she was totally blind.

In addition, there was some proptosis of the right eye, together with an ophthalmoplegia which then developed. Late in the course of the illness marked menstrual irregularities with amennorhea occurred, probably as result of the intracranial extension and pituitary gland involvement.

X-rays of the skull revealed progressive destruction of the sella turcica to the point that it disappeared almost completely within the expanding tumor mass.

Post Mortem Findings: Complete post mortem examination revealed no evidence of metastatic spread of the primary tumor. The

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tumor mass was found to involve both sides of the nasal cavity as well as the septum. It grew upward and backward eroding through the base of the skull to protrude into the anterior and middle fossa of the skull. The sella turcica was completely destroyed as were the anterior tips of the petrous portions of both temporal bones. The optic chiasm was stretched upward and backward over this expanding mass of tumor and the right optic nerve disappeared into the tumor mass. Antero-posteriorly the tumor extended from the middle of the cribriform plate of the ethmoid to the anterior margin of the foramen magnum.

SUMMARY AND CONCLUSIONS

Tumors of cartilaginous origin arising within the nose or paranasal sinuses are uncommon. They may be histologically benign enchondromas, or malignant chondrosarcomas. However, their very location near vital structures in the head make almost all such tumors clinically malignant. Their usual course is a slowly progressive one with persistent growth and expansion, gradually eroding normal bone about them and often invading the intracranial cavity.

We have had the unusual opportunity to observe the course of a patient who developed a chondrosarcoma which arose intranasally and, ultimately, extended into the cranial cavity. We have been able to correlate the developing clinical and x-ray findings with the actual pathological changes as observed at surgical exploration and at the postmortem examination.

The diagnosis of cartilaginous intranasal tumors is established by biopsy. Many show a relatively benign histologic appearance but certain criteria for determining malignant change have been suggested. Since their location within the nasal cavity makes the benign enchondromas as potentially dangerous as the chondrosarcomas, it would seem that the same treatment should be applied to all intranasal tumors of cartilaginous origin.

In the vast majority of cases, such tumors are radioresistant, responding only to a limited degree to radiation therapy. Most reports indicate that the treatment of choice is surgical excision of the tumor mass. Good exposure of the tumor developing within the nasal cavity or from the ethmoid region and projecting into the nasal cavity may be obtained by means of the Fergusson incision or a modification of it. Modern methods of anesthesia, good preoperative preparation, and the availability of whole blood will permit adequate exploration of these tumors and wide excision when feasible.

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SCIENTIFIC PAPERS OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION

(Continued from June issue)

LXIX

THE INDICATIONS AND TECHNIQUE FOR CERVICAL LYMPH NODE RESECTION

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Intra-oral cancer is of extreme importance because of its frequency, the ease of early detection, and the fact that it is generally amenable to therapy. According to Hayes Martin,[11] cancer of the mouth represents 70 per cent of all cancers in the upper alimentary and respiratory tracts and five per cent of all cancers. It is primarily a disease of men. Eighty to 90 per cent of patients with carcinoma of the tongue, gums, tonsils, palate, and cheeks, and 98 per cent of patients with carcinoma of the lip and floor of the mouth are males.[11]

Because of the rich lymphatic drainage from the oral cavity, lymph node involvement from lesions in the buccal cavity is extremely likely. Involvement of the lymph nodes is undoubtedly favored by the extremely rich lymphatic drainage and also the mobility of the area in which the primary lesion is located, both of which tend to favor extension through the lymph stream. Braund and Martin[4] found cervical node metastasis at autopsy in 23 per cent of a group of patients with carcinoma of the mouth, pharynx, larynx, and esophagus. Although lesions of the oral cavity metastasize frequently, and usually quite early, to the cervical lymph nodes, it is of importance that the cervical lymphatic system is a relatively closed system and that distant metastases are not likely to occur (Figs. 1 and 2). Although most tumors of the buccal cavity, when they metastasize to the cervical nodes, usually remain localized in the cervical nodes indefinitely, there are two types of tumors in which widespread metastases are likely to occur. These are the transitional carcinomas and the lympho-epitheliomas which are found at the base of the tongue and in the region of the tonsils. In Duffy's[6] series of cases, 20 per cent of lesions at the base of the tongue were

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either transitional carcinoma of lympho-epithelioma. According to Boyd[3] lympho-epitheliomas or transitional cell carcinomas represent 10 per cent of malignant tumors of the mouth and pharynx. According to Martin and Blady,[12] 34 per cent of their patients with nasopharyngeal carcinoma had distant metastases on admission. These were primarily to the liver, lungs, mediastinal and abdominal nodes, and the lumbosacral area.

The indications for cervical lymph node dissection are dependent upon a number of factors. Obviously a lymph node dissection is not indicated unless the primary lesion has been cured or there is chance of its being cured, because the only reason for doing a lymph node dissection is in an attempt to cure. If the primary lesion has been eradicated or if there is a likely chance that it can be eradicated and there is obvious involvement of the cervical nodes, lymph node dissection is indicated. There is considerable controversy concerning the advisability of performing prophylactic lymph node dissection, that is, removal of cervical lymph nodes when there is no clinical evidence of their involvement. It must be emphasized, howeevr, that the finding of enlarged lymph nodes does not necessarily mean involvement with carcinoma, because most malignant lesions of the oral cavity are infected and microorganisms as well as malignant cells can be carried through the lymph channels. Lymphadenopathy may be the result of an inflammatory enlargement. Blair et al[2] found that in their cases with clinically enlarged nodes no tumor was found microscopically following lymph node dissection in 35 per cent. Similarly, Phillips[18] found that of 59 cases with lymph node enlargement which appeared clinically to be malignant no carcinoma was found in 22 (37 per cent). On the other hand, it is a well-known fact that the absence of palpable lymph nodes is no proof that the lymph nodes are not involved. Kennedy,[9] in a series of patients with carcinoma of the lip with no clinical evidence of lymph node involvement, found microscopic evidence of involvement in 14 per cent. Martin[10] found in 40 patients with carcinoma of the tongue with no palpable nodes that 12 per cent subsequently developed cervical metastases and that of 12 patients with carcinoma of the cheek with no palpable nodes, 17 per cent subsequently developed cervical metastases. Morrow[17] found in a series of cases of carcinoma of the tongue that there was involvement of the cervical nodes in one-third of the cases in which there was no clinical evidence of lymph node involvement. Phillips,[18] in a series of 31 cases of buccal carcinoma in which

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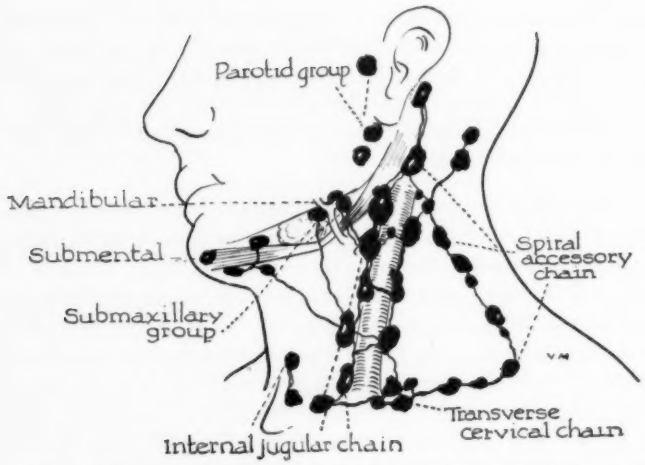

Fig. 1.—Diagram of lymph nodes of the cervical region, which may be involved in lesions of the mouth and pharynx. Radical neck dissection consists of removal of all the nodes shown in this drawing.

there was no clinical evidence of metastases, found that 16 (51 per cent) had microscopic involvement. Roux-Berger and Tailhefer,[20] in a series of 494 cases of carcinoma of the mobile portion of the tongue in which bilateral neck dissections were done, had 18 cases with no palpable nodes but in all of these the nodes were involved microscopically. Simmons[21] found cervical node involvement on microscopic examination in 34 per cent of his patients, although there was no clinical evidence of involvement.

As mentioned previously, there is no uniformity of opinion concerning the advisability of performing prophylactic lymph node dissection. Brown et al,[5] Ward and Hendrick,[25] and Willy Meyer[16] are advocates of prophylactic dissection. Martin et al[13] are opposed to prophylactic dissection. In a series of 237 consecutive cases with cancer of the lip treated at the Memorial Hospital, Martin et al found that if prophylactic dissection had been done, it would have been of value in only 6.25 per cent and that "fifteen useless operations would have been necessary in order to have been of benefit in one case." Similarly, in 120 cases of cancer of the tongue, prophylactic

dissection would have been of benefit in 20 per cent or "four useless operations would have been necessary in order to be of possible benefit in one case." In a questionnaire dealing with carcinomas about the mouth which they sent to 75 persons, 46 per cent replied that they would not perform prophylactic neck dissections either for cancer of the tongue or the lip, 75 per cent were opposed to prophylactic neck dissection for cancer of the lip, 17 per cent thought it was indicated in all cases of lingual cancer, and 10 per cent thought it should be done in all cases of lip cancer. There are many who believe that in lip cancer, careful observation of the patient can be done in order to determine whether lymph node involvement occurs or not because lip cancer is relatively slow-growing and because metastases, when present, are usually limited to the submaxillary and submental group of nodes. Such a plan of therapy is possible only if frequent examinations of the patient are possible, at least every three months for the first 18 months and then every six months for several years. Figi[8] believes that the risk of prophylactic lymph node dissection is greater than the benefit of the procedure. On the other hand, Willy Meyer[16] advocates prophylactic suprahyoid dissection in all lip carcinomas and states that in 14 per cent of their patients with no palpable nodes there was microscopic involvement of the nodes. He also found 39 per cent microscopic lymph node involvement in lesions of the tongue although there was no clinical evidence of the involvement. He is of the opinion that since there is a decussation of the lymphatics along the median raphe of the tongue that "in every tongue carcinoma the cancer field includes the entire lymphatic field on both sides of the neck. If you want to try to arrest the disease, you have to do a bilateral lymph node dissection and that dissection has to go from the mandible down to the clavicle on both sides, because the lymphatics of the tongue decussating go through the hypoglossal muscles and can go right along the omohyoid muscle into the supraclavicular fossa, skipping the nodes that lie over the jugular vein area."

It is our opinion that prophylactic lymph node dissection is necessary in many cases, but is not necessary in cancer of the lip unless the lesion is large, that is, greater than 3 cm or is highly malignant. There is considerable variation in the rate of metastases according to the location of the malignant lesion, the degree of malignancy, and its size. In Taylor and Nathanson's[24] series of lip carcinoma, metastases occurred as follows: in lesions less than 1 cm,

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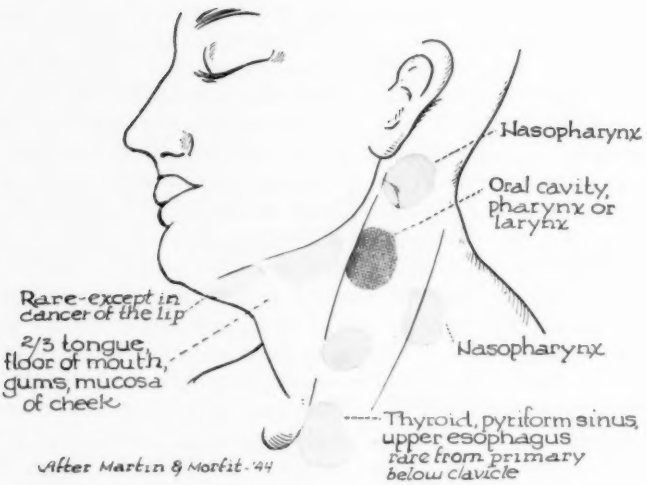
Nasopharynx
Oral cavity, pharynx or larynx
Rare-except in cancer of the lip
2/3 tongue, floor of mouth, gums, mucosa of cheek
Nasopharynx
Thyroid, pyriform sinus, upper esophagus
rare from primary below clavicle
After Martin & Morfit-'44

Fig. 2.—Diagram illustrating the usual sites of metastasis with the primary lesions designated. (Redrawn after Martin and Morfit)

7 per cent, from 1-2 cm, 13 per cent, 2-3 cm, 37 per cent, and over 3 cm, 24 per cent. They also found that lymph node metastases were dependent upon the degree of malignancy: Grade I, 6 per cent metastases, Grade II, 30 per cent metastases, Grade III, 52 per cent metastases. Similar results were found in carcinoma of the buccal mucosa. Lesions less than 1 cm had 15 per cent metastases, from 1-2½ cm, 34 per cent metastases, from 2½-4 cm, 37 per cent metastases, over 4 cm, 44 per cent metastases. Lesions of Grade I had 24 per cent metastases and Grades II and III, 45 per cent metastases.

Although cancer of the lip is the most frequent cancer involving the buccal cavity (25 to 30 per cent)[1] and in fact is the most frequent cancer of the upper respiratory and alimentary tracts, lymph node dissections are necessary and are done more frequently for cancer of the tongue. In a series of 500 consecutive cases of cervical metastatic cancer reported by Martin et al,[14] the lip was the site of the original lesion in only seven per cent, the tongue was the site of the original lesion in 25 per cent, the floor of the mouth in 9 per cent, the nasopharynx in 8 per cent, the mucosa of the cheek in 6 per cent, the

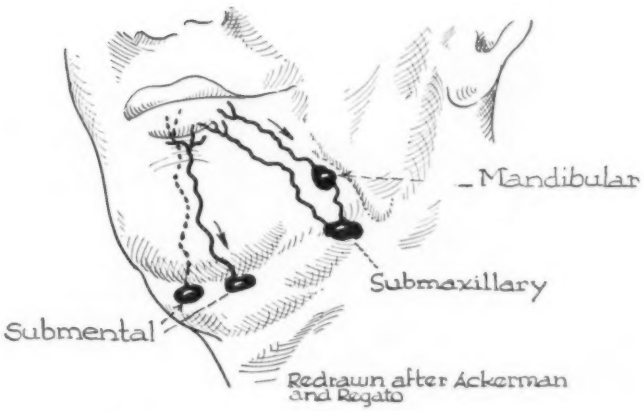

Fig. 3.—Diagram illustrating lymphatic involvement from lesions of the lower lip. The submaxillary nodes are most frequently involved. Occasionally a mandibular node is involved and rarely the submental if the lesion is in the midportion of the lip. (Redrawn after Ackerman and Regato)

jaws in 5 per cent, the pharyngeal wall in 5 per cent, and the palate in 4 per cent. It is thus seen that although cancer of the lip occurs more frequently than cancer of the tongue, metastases to the lymph nodes in the latter occur much more frequently. As mentioned previously, we are of the opinion that lymph node dissection in cancer of the lip is not necessary, provided there are no palpable nodes and provided the lesion is smaller than 3 cm and not anaplastic. In recurrent lesions of the lip which have been treated previously, it is desirable that at least a suprahyoid resection should be done. It is of importance, however, that the patient in whom a prophylactic node resection is not done, should be followed regularly at least every three months for one-and-one-half years, then every six months for five years, and then once a year for the rest of his life. Even though microscopic involvement of the lymph nodes can occur in carcinoma of the lip without clinical evidence, we believe since these lesions remain localized for a relatively long period of time that by careful follow-up examination a metastatic lesion can be detected at a time when it is still curable. The lymph nodes which are likely to be involved in cancer of the lip vary according to the location of the tumor. The lymph nodes involved in the drainage from the lower

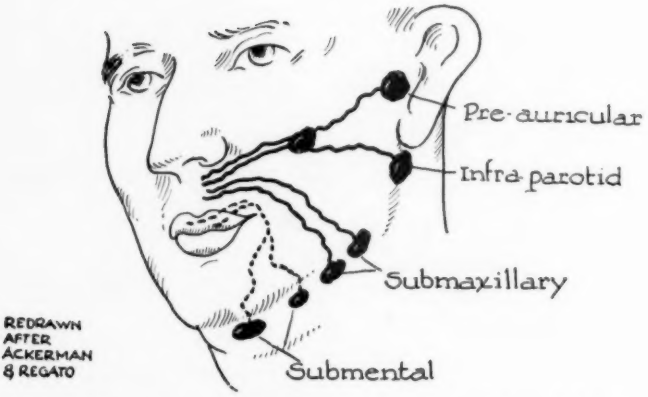

Fig. 4.—Diagram showing lymphatic drainage from lesions of the upper lip. Although lymph node metastases from lesions of the upper lip are much less frequent than those of the lower lip, they are likely to be widespread as illustrated in the diagram. (Redrawn after Ackerman and Regato)

lip are principally the submaxillary, mental, and submental nodes. (Fig. 3) When the lesion is near the midline there is involvement of the nodes at the mental mandibular foramen and occasionally the superior deep cervical nodes. Careful examination of all these areas should be made in any patient who has or has had a carcinoma of the lip. In carcinoma of the upper lip, involvement may be the preauricular nodes, the infraparotid, the submaxillary, the submental, and the deep cervical chain. (Fig. 4) In contradistinction to carcinoma of the lower lip, a malignant lesion of the upper lip is frequently associated with widespread lymph node involvement. Martin et al found 48 per cent of lymph node metastases in 21 cases of upper lip carcinoma.

The type of lymph node dissection for carcinoma of the lip varies somewhat according to the location of the lesion, the presence or absence of metastases, and the grade of malignancy. In large lesions or anaplastic lesions without evidence of lymph node involvement, a unilateral suprahyoid resection is indicated if the lesion is located on the lateral part of the lip. A bilateral suprahyoid resection is done for lesions in the midline. In the presence of suprahyoid involvement, a unilateral radical neck dissection on the involved side

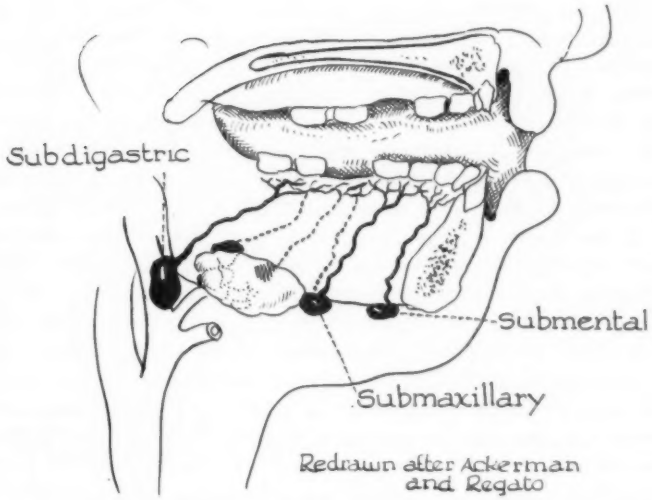

Fig. 5.—Diagram illustrating lymph node metastases from lesions of the lower gingiva. There is common involvement of the lymph nodes. Most frequently involved are the submaxillary group, the external maxillary group, occasionally the submental, and not infrequently the superior cervical nodes are secondarily involved. (Ackerman and Regato)

is done or a bilateral dissection at different stages if the lesion is near the midline. A suprahyoid resection carries much less risk than a radical neck dissection and can be done with a considerable degree of safety.

Carcinoma of the gingiva is usually a relatively benign lesion, but lymph node involvement varies according to location of the carcinoma. Those cancers involving the lower gingiva are likely to be associated with lymph node metastasis much more frequently than upper gingival lesions due to proximity of the lesion to the lymphatics on the outer surface of the mandible and the frequency of involvement of the floor of the mouth. (Fig. 5) When the lymphatic channels on the external surface of the mandible are involved, one can frequently feel thickened cords on the external surface of the lower jaw. Also the lesion is relatively close to the floor of the mouth and can, if neglected, extend to this area from which there

is a greater likelihood of metastatic extension than if it had remained limited to the gingiva. Taylor and Nathanson[24] found 65 per cent of their cases with carcinoma of the lower gingiva had lymph node metastasis. In each instance, however, there was involvement of the adjacent cheek, the floor of the mouth, or the fauces. They also found that lymph node metastasis varied according to the location on the lower jaw as follows: anterior one-third, 50 per cent, middle one-third, 60 per cent, posterior one-third, 66 per cent, the entire side, 70 per cent. Lymph node metastasis was highest when the fauces or floor of the mouth was involved also, 77 per cent, whereas when the gingiva and cheek were involved, 55 per cent had metastases. When the gingiva alone was involved, 48 per cent had metastases. Because of the proximity of carcinoma of the gingiva to the mandible, there can be direct extension to and involvement of the mandible. This is of importance as regards eradication of the original tumor, because in all cases of gingival carcinoma in which the lesion is large, it is necessary that the adjacent portion of the mandible should be included in the resection. The lymph nodes which are usually involved are the submaxillary group, the external maxillary group, occasionally the submental, and the superior deep cervical nodes.

The treatment of carcinoma of the lower gingiva, unless the lesion is small and superficial in which a wide location excision can be done, should consist of en bloc excision of the lesion, the adjacent mandible, and radical neck dissection on the homolateral side.

In contradistinction to carcinoma of the lower gingiva, carcinoma of the upper gingiva is associated with lymph node involvement infrequently and when present usually occurs late. The lymph nodes which are involved are the submaxillary nodes, seldom the superior cervical nodes, and relatively late, the jugular nodes. (Fig. 6) Although the lesion is a relatively rare tumor, it can metastasize and should be treated adequately with wide excision of the local lesion with radical neck dissection on the homolateral side, if palpable nodes are present and if the tumor is a large one. Carcinoma of the hard palate is very similar to carcinoma of the upper gingiva and can be treated in approximately the same way.

Carcinoma of the tongue is somewhat more frequent in women than other carcinomas of the mouth. Approximately one-fifth of the carcinomas of the tongue are found in the female sex. Although carcinoma of the tongue occurs much less frequently than carcinoma

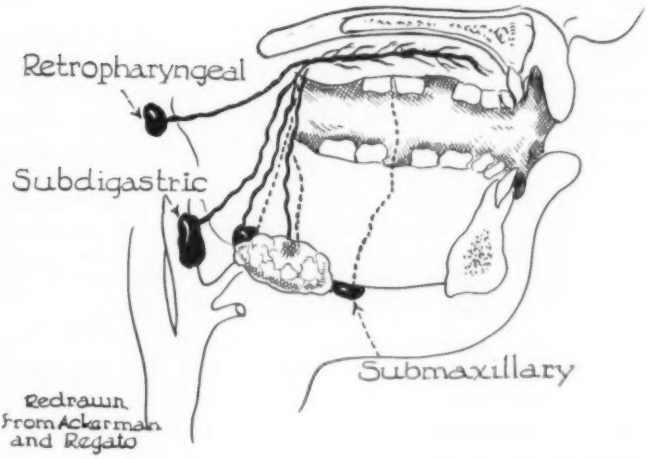

Fig. 6.—Diagram showing lymph node drainage in lesions of the upper gingiva. Although lymph node involvement of lesions of the upper gingiva are rare, when they do occur they are to be the submaxillary nodes and may extend to the superior cervical nodes. Rarely they extend to the retropharyngeal area. (Redrawn after Ackerman and Regato)

of the cheeks, lips, and lower gingiva, lymph node metastases occur much more frequently. In the cases reported by Sommervell[22] the tongue was involved in only 13 per cent, whereas the cheeks and lips were involved in 52 per cent, the lower jaw in 28 per cent, and the upper jaw in 7 per cent. Martin, Munster, and Sugarbaker[15] found that 35 per cent of their patients had lymph node metastases when admitted and 25 per cent subsequently developed them, giving a total of 60 per cent that had lymph node metastases and only 40 per cent who did not. They found that in 30 per cent of their cases with cancer of the posterior part of the tongue, the first manifestation was cervical lymphadenopathy. Thirty-nine per cent of their cases had, on admission, gross evidence of lymph node involvement and subsequently 24 per cent developed it, making a total of 63 per cent who had lymph node metastases. Of 500 consecutive cases with lymph node metastases reported by Martin et al,[13] the originating lesion was the tongue in 25 per cent. Morrow[18] found that in one-third of his cases with cancer of the tongue in which there were no

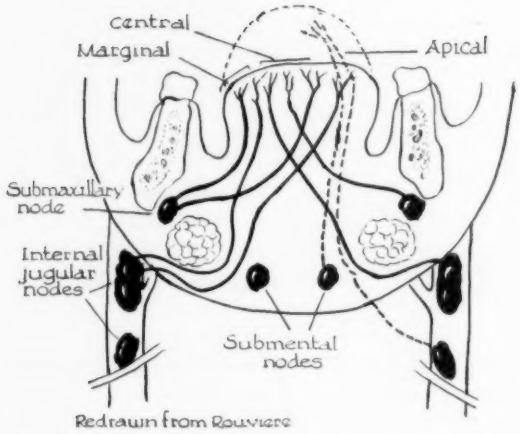

Fig. 7.—Diagrammatic drawing showing lymphatic drainage from the tongue. The lymphatic drainage from the tongue depends to a certain extent upon the lesion, whether it is at the tip, the base, the lateral border or the median portion. The superior cervical node or the subdigastric node is very frequently involved and is frequently described as "node of the tongue." As shown in the diagram, lesions of the tongue because of decussation of the lymphatic drainage, can metastasize to the opposite side. Lesions of the tip of the tongue can extend to the submental nodes. (Redrawn after Rouviere)

palpable lymph nodes tumor was found in the nodes microscopically. In 494 cases of carcinoma of the mobile portions of the tongue reported by Roux-Berger and Tailhefer,[21] bilateral neck dissections were done in 18, none of which had palpable nodes, but cancer was found microscopically in all 18. In 306 cases of cancer of the anterior two-thirds of the tongue in which the lesion was strictly unilateral, 6 per cent had bilateral metastases. In 188 cases in which the lesion extended beyond the midline, 32 per cent had bilateral metastases. Sugarbaker[23] found metastases to the cervical lymph nodes in 63 per cent of his patients with carcinoma of the tongue and floor of the mouth. As evidenced by these statistics, malignant lesions of the tongue, particularly the mobile portions, are particularly likely to metastasize to the regional lymph nodes. (Fig. 7) This is undoubtedly due to several factors: 1) the extreme mobility of the tongue which tends to massage the tumor emboli through the lymphatics, 2) the

rich lymphatic drainage of the tongue, and 3) the fact that there is bilateral lymphatic drainage from the tongue. Lesions of the anterior two-thirds of the tongue are generally epidermoid lesions, but approximately 20 per cent of lesions of the base of the tongue are transitional cell carcinoma or lympho-epithelioma. In transitional cell carcinoma and lympho-epithelioma, there is a great tendency to systemic involvement.

Lymph node drainage of the tongue should be divided into four areas: the tip, the lateral border, the base, and the median portion. Lymphatics at the apex or tip of the tongue course posteriorly and downward. Taylor and Nathanson[24] found lymph nodes involved in 71 per cent of their patients with carcinoma of the tongue. The principal node involved is the subdigastric node, which is often described as the node of the tongue. According to Morrow[17] the tip of the tongue is involved in 4.3 per cent of cases, the anterior two-thirds of the margin in 43.5 per cent, the posterior one-third in 16.3 per cent, the dorsum in 14.1 per cent, the base in 5.4 per cent, the undersurface in 10.8 per cent, and the whole tongue in 5.4 per cent. From the tip, the anterior jugular chain is involved. As emphasized by Figi,[8] the more anterior the lesion, the lower in the jugular chain is the involvement likely to be. As mentioned previously, because of the bilateral lymph drainage of the tongue, contralateral metastases are likely to occur. (Fig. 7) In Martin et al series,[13] in lesions of the anterior two-thirds, metastases crossed the midline in 21 per cent. In lesions which were distinctly unilateral and subsequently developed cervical metastases, 10 per cent appeared contralaterally at first and lymph node metastases occurred bilaterally in 24 per cent of the cases. There was a possible bilateral involvement in 32 per cent. Metastases from the lateral border of the tongue are to the submaxillary lymph nodes, the superior deep cervical nodes, and the deep cervical chain. As mentioned previously, the more anterior the location of the original lesion, the deeper the involvement of the cervical chain. (Fig. 8) Lymph node involvements from the base of the tongue are primarily to the superior cervical nodes; cervical node involvement is frequently the first evidence of disease. This is followed in frequency by involvement of the deep cervical chain. Bilateral involvement is likely to occur in such an instance. Lymph node involvement from the median portion of the tongue is almost invariably followed by bilateral lymph node involvement to the submaxillaries, superior deep cervical, and the cervical chain.

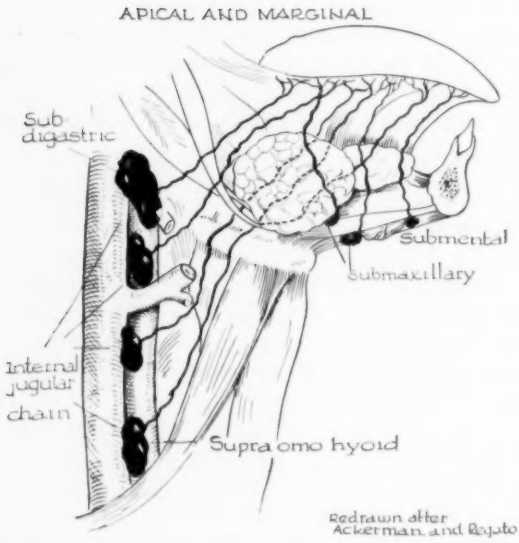

Fig. 8.—Diagrammatic drawing showing lymphatic drainage of the marginal and apical portions of the tongue. Drainage is primarily to the subdigastric or superior cervical nodes, but drainage can extend down to the deeper portions of the jugular lymph node group and generally the more anterior the lesion the more inferior in the internal jugular group is the nodal involvement. (Redrawn after Ackerman and Regato)

Because of the immediate attachment of the tongue to the floor of the mouth, lesions of the tongue can be considered together with those of the floor of the mouth. A lesion of the tip of the tongue can be treated by wide excision followed by bilateral suprahyoid dissection with unilateral neck dissection if more on one side. The treatment of cancer of the lateral border of the tongue and involvement of the floor of the mouth consists of irradiation followed by en bloc excision and radical neck dissection on that side or a radical en bloc hemiglossectomy and cervical block dissection. The treatment of cancer of the inferior surface of the tongue with involvement of the floor of the mouth consists of radical en bloc excision of the tongue lesion, the floor of the mouth, the adjacent mandible, and all the cervical nodes. If the lesion is near the midline, the same

procedure must be done, but later radical neck dissection should be done on the opposite side. The treatment of carcinoma of the base of the tongue consists of irradiation followed by radical neck dissection on the homolateral side and bilateral radical neck dissection if the lesion is near the midline.

Carcinoma of the soft palate which frequently involves the faucial pillars is fortunately a rare condition. It is extremely rare in women. It is associated with early metastasis due to rich lymphatic drainage, the muscular activity of the palate which undoubtedly aids in the movement of metastatic emboli. There is frequently bilateral lymph drainage. Because of its high degree of malignancy, lymph node metastases occur in a large percentage of cases. The nodes which are involved are the superior cervical nodes, the submaxillary nodes, and the retropharyngeal nodes. Because of the fact that there is early lymph node metastasis and because it extends widely and frequently bilaterally and to the retropharyngeal area, the prognosis is bad and very few cures can be obtained. It is of importance to radicate completely the local lesion either by irradiation or surgical extirpation. If this has been accomplished satisfactorily, a bilateral radical neck resection should be done.

Cancer of the buccal mucosa of the cheek is a relatively benign lesion except when it is located posteriorly near the angle of the jaw in which case it is usually much more malignant. In 98 cases reported by Duffy,[6] there were lymph node metastases in 60 per cent. Martin[11] found that in 12 patients with carcinoma of the cheek with no palpable nodes, 17 per cent subsequently developed lymph node metastases. Richards[19] has emphasized the relative benignancy of carcinomas of the buccal mucosa and states that they remain localized in from 50 to 60 per cent and that when they do extend they extend to the submaxillary gland and remain there. They do not extend beyond the submaxillary gland as rapidly and as far as do carcinomas of the tongue and the floor of the mouth. The lymph nodes which are involved in carcinoma of the cheek are usually the submaxillary and the superior deep cervical nodes. Although the involvement varies according to the location of the lesion, lesions located in the anterior portion of the cheek extend to the submaxillary nodes and the submental nodes. Those lesions which are located in the posterior portion of the cheek extend to the parotid nodes and the superior deep cervical nodes. It is imperative that the local lesion should be recognized early and treated adequately. This can usually be

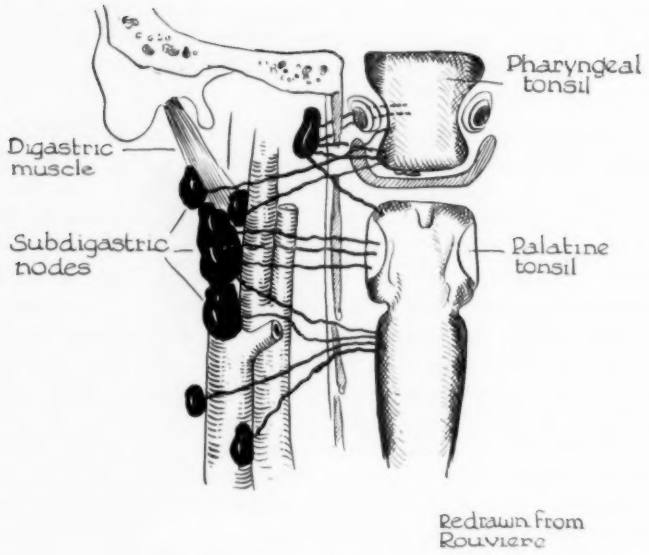

Fig. 9.—Diagrammatic representation of the lymph drainage from the pharynx. The drainage is to the internal jugular nodes, particularly the subdigastric nodes, the superior cervical nodes, and the retropharyngeal nodes. (Redrawn after Rouviere)

done satisfactorily by irradiation, but it should be followed by a unilateral radical lymph node dissection, because the lesion frequently extends to the deep cervical nodes. It is necessary in doing a lymph node dissection to do a much more radical excision than a suprahyoid dissection. It is for this reason that a radical lymph node dissection is indicated.

Cancer of the tonsil is a frequent lesion and according to Ewing[7] represents 10 per cent of all buccal cancers. Malignant lesions of the tonsil are usually lympho-epithelioma, transitional cell carcinoma, or lympho-sarcoma. One-third of the lympho-epitheliomas and lymphosarcomas occur in women.[1] Squamous cell carcinomas are extremely rare. Lymph node involvement is frequent and early and may be the first clinical manifestation. This is due to the rich lymphatic drainage of the tonsil, the fact that there is bilateral lymph

drainage, and that the lesions are highly malignant. The nodes which are usually involved are the superior deep cervical nodes, the deep cervical chains, and the subparotid nodes. The treatment consists first of irradiation which should be done intensively and, if the lesion is a lymphosarcoma, usually good palliative results will be obtained. However, at the present time, there is an increasing tendency to do combined operations which consist of an en bloc excision of the original lesion and the unilateral radical neck dissection after irradiation of the primary lesion has been done. In this way, the primary lesion is shrunken and the operation can be done much more satisfactorily. If the lesion is close to the mandible, it is necessary to resect that portion of the mandible. On the other hand, the mandible may be divided and subsequently sutured in order to complete the operation.

Cancer of the pharynx is usually a highly malignant tumor and may be either epithelial or mesothelial in origin. According to Martin and Blady,[12] 34 per cent of their patients with nasopharyngeal carcinoma had distant metastases at the time of admission. These metastases were to the liver, lungs, mediastinal and abdominal nodes, and the lumbrosacral area. Sugarbaker[23] found positive lymph node metastasis in 80 per cent of his cases of carcinoma of the nasopharynx. The lymph node involvement is early due to the rich lymphatic drainage in the region of the pharynx and the highly anaplastic lesion. The lymph nodes which are involved are the superior deep cervical, the subparotid, and the deep cervical chain. The treatment consists of intensive irradiation, lymph node dissection seldom being indicated.

Radical lymph node dissection of the neck, which usually should be done when lymph node resection is indicated, is an operation which must be done carefully and meticulously. It consists of the removal of all the areolar, lymphatic, and fatty tissues together with the inferior pole of the parotid, and the external and internal jugular veins, the sternocleidomastoid muscle from the clavicle up to the mandible. It is a tedious procedure, but must be done carefully if good results are to be obtained. It is done as an en bloc dissection and the block of tissue contains the submental nodes, the submaxillary nodes, the submaxillary salivary gland, the inferior pole of the parotid, the deep cervical nodes, the digastric nodes high in the neck, in addition to the entire sternocleidomastoid, internal jugular vein, the omohyoid muscle, and the posterior belly of the digastric muscle and the sterno-

hyoid muscle. The vagus, the phrenic, the hypoglossal and lingual nerves, and the carotid artery are left intact. If there is any apparent involvement of the spinal accessory nerve, this should be sacrificed, although many times it can be spared.

In many instances, particularly in lesions of the gingiva and the floor of the mouth and tongue, it is desirable to do a combined operation which consists of an en bloc excision of the horizontal ramus of the mandible, the intra-oral lesion, and the cervical lymph nodes. The removal of the mandible is essential because it has been shown that in a high incidence of approximately 50 per cent of instances, lymphatics pass through the periosteum of the mandible.

Suprahyoid dissection consists of the removal of the subdigastric nodes, the submaxillary nodes, including the submaxillary gland, the submental nodes on both sides, the removal of all the fat, areolar tissue and lymphatic tissue in the submaxillary triangle. This may be done unilaterally or bilaterally.

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LXX

PROGRESS IN LARYNGOFISSURE

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Thyrotomy or laryngofissure is one of the oldest surgical procedures in the repertoire of the present day laryngologist. Strangely enough, this operation, which has come to be associated with carcinoma of the larynx, was first employed for removal of a foreign body. It was not until 1851 that it was used as an approach to the surgical treatment of laryngeal carcinoma. The procedure received increasing attention in the early twentieth century but so few reports appeared in the literature that for a time its value was questioned. During this period the literature was absorbed with discussions of the surgical treatment of virulent infections, which were so prevalent. However, antibiotics have now controlled this problem and during the past ten years attention has been focused upon preventive medicine, control and cure of chronic infections and cancer. During this time the laryngofissure operation has become increasingly popular. It has proved to be an accepted operative procedure of unquestionable value not only in the arrest of cancerous growths of the larynx but also in the conservation of speech in these unfortunate patients.

As our technique has improved with experience, we have broadened our indications for the procedure. The original indication for laryngofissure was a lesion limited to the cordal level with good motion of the cord (Fig. 1). This is still considered the ideal type of case, and we have obtained the highest incidence of cure (86 per cent) in this group. Eighty per cent of these had Grade I or Grade II carcinoma, the remainder having the higher grades. Of the 1.4 per cent recurrences in this group, 85 per cent were Grade III or IV carcinomas.

It has been shown that the higher the degree of malignancy the further the spread of the lesion beyond the gross evidence of invasion. Therefore, in the past eight years we have performed much wider

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Fig. 1.—Ideal case for laryngofissure, and in low-grade carcinomas, for intralaryngeal extirpation.

Fig. 2.—Malignant neoplasm of left vocal cord crossing the midline at the anterior commissure and extending to the vocal process. The cord alone is involved and motion is good. Although laryngofissure is usually considered contraindicated in this type of lesion, good results have been obtained in Grade I and Grade II carcinomas following extensive laryngofissure and cauterization.

excision of tissue and much more extensive cauterization than formerly. As a result of this the percentage of cures to date has been even higher (90.5 per cent). The 10 per cent recurrences in this group still are predominantly in the Grade III and Grade IV types. However, in the entire series the incidence of recurrences is not nearly this high, the reason being that in the majority of instances in which the lesion has crossed the commissure anteriorly but in which there is still good movement of the cords (Fig. 2) laryngofissure may still be done successfully. Broyles[1] has shown that if the lesion approaches the anterior commissure, often it extends beyond the insertion of the conus to invade the cartilage in small islands. Therefore, in lesions involving the commissure a section of the thyroid cartilage anteriorly surrounding the insertion of the conus must be removed. This has enabled us to do more extensive laryngofissures than formerly. Our results have warranted widening the field of indications by being radical and yet at the same time we have been able to conserve a functioning larynx.

If the anterior commissure is disturbed, and this is especially true in crossing the midline to remove a portion or all of the opposite cord, a web will result. Previous attempts at correction of this web have met with varying degrees of success. However, we have now adopted the technique of McNaught[2] in which a tantalum splint (Fig. 3) is inserted between the scarred tissues, which is nature's attempt to regenerate a cord. This requires two surgeons. One operator sitting at the head and using the suspension laryngoscope first removes the excess scar tissue up to the anterior commissure. The second surgeon then reopens the larynx but through a much smaller incision than the original laryngofissure incision. The opening is made first long enough to insert the tantalum splint. The first operator then guides this splint into the correct place, being sure it separates the tissues in the midline and that it is not too long so that there is no contact with the posterior wall of the larynx. This is usually removed in six to eight weeks under local anesthesia as an ambulatory case. We have found it unwise to insert this splint at the time of the original laryngofissure procedure because of the tendency to excessive granulation tissue as a result of this foreign body. However, six to eight months after operation when a firm web has formed, the extent of scar to be removed, and most importantly, the amount to be left for a good substitute cord, can then be accurately determined.

Fig. 3.—Roentgenogram showing laryngeal splint in place.

Our indications have been extended still further to include cases with early partial limitation of motion (Fig. 4) due to extension of the growth beyond the caudal line laterally or posteriorly to the attachment of the cord at the vocal process. However, once the arytenoid itself is involved or the growth is completely or almost completely fixed, laryngofissure is contraindicated. With limited motion by extension laterally subperichondral dissection is done, which incidently is done in all cases. The false cord ventricle and subglottic tissue to the lower border of the thyroid cartilage may be removed in this manner. Following removal by sharp dissection, the raw surfaces superiorly, inferiorly and posteriorly are subjected to extensive cauterization with the Bovie unit. If, however, the growth extends to the vocal process (Fig. 5), not only is the entire cord removed but also a portion of the arytenoid cartilage, followed

again by extensive cauterization. Cauterization in this area causes considerable reaction, and usually for the first two or three days patients are fed intravenously or by means of a Levine tube, as they have extreme difficulty in swallowing due to the reaction as well as pain. The laryngofissure operation may be carried even further with the recent procedure presented by Figi[3] in which the ala of the thyroid cartilage may also be removed followed by skin grafting.

In none of these procedures has tracheotomy been necessary, either preliminary to or in conjunction with laryngofissure. The laryngofissure operation is performed under local anesthesia. Elimination of the tracheotomy has resulted in considerable reduction in the duration of postoperative hospitalization, the average time in the hospital being from five to seven days.

Complications of laryngofissure have been minimal. Emphysema is, of course, the commonest, because no tracheotomy is performed. It develops in only about 10 per cent of cases. The secret of preventing development of this complication is tight closure of the cricothyroid membrane. However, as our scope of indications has widened to include cases in which a portion of the anterior border of the thyroid cartilage has been removed, slight dehiscence remains. Tight closure of the perichondrium plus use of an elastic bandage prevents development of emphysema in many cases. In these cases a small twisted dermal drain is inserted and a direct pathway for the escape of air is established if this should occur. Emphysema develops superficial to the superficial fascia, may extend to the chest or even up to the eyes but does not embarrass respiration, and even when extensive, disappears by the third or fourth day. Secondary hemorrhage is exceedingly rare, having occurred in only two of our cases. Both times tracheotomy was necessary. In one the larynx could be packed following tracheotomy and general anesthesia but in the second the wound had to be reopened and the bleeding vessel ligated. Both of these occurred from one to two weeks postoperatively when the slough of the cauterized tissue was separating. In only one case was tracheotomy indicated postoperatively and this resulted from radical removal and more extensive cauterization producing secondary edema of the arytenoids.

In the treatment of carcinoma of the larynx we employ either laryngofissure or laryngectomy. With experience the indications for the former have gradually been broadened. Figi[3] and Pressman[4]

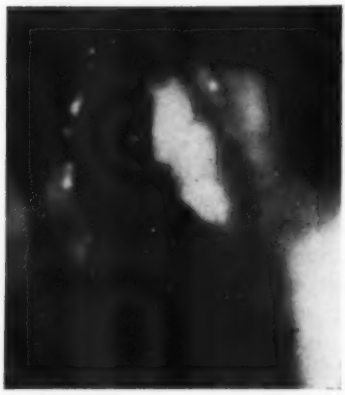

Fig. 4.—Borderline lesion with involvement beyond the cord and limited movement of the cord. Wide laryngofissure and cauterization even in Grade III carcinomas give good results.

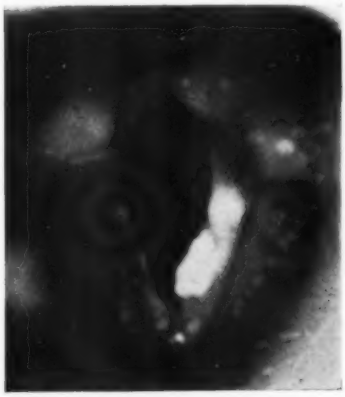

Fig. 5.—More advanced case with involvement of the vocal process necessitating partial removal of the arytenoid. Following laryngofissure an adequate airway is provided but a poor voice results.

have devised procedures to widen the scope even more in attempting to maintain an adequate airway and a functioning larynx. The wider the scope of laryngofissure, the more closely one approaches the line of demarcation between this procedure and total removal of the larynx. Therefore, these patients have to be watched more closely and for a longer period of time than those who have had less radical operations. Naturally, the incidence of recurrence is greater. However, in view of the fact that 73 per cent of our patients have not had a recurrence, this means that many more patients have been spared loss of their voice and laryngeal airway. In the remaining 27 per cent of recurrences, a further laryngofissure procedure is not attempted; laryngectomy is then the operation of choice. When these patients have returned for frequent periodic examinations, arrest of the disease has been obtained in 85 per cent of the recurrences.

Since the original laryngofissure operation, progress has been phenomenal. We are gradually approaching the possibility that in the majority of patients with cancer of the larynx adequate removal of the tumor can be accomplished without destroying the function of the larynx. This possibility, however, can be achieved only by more widespread education and preaching of the doctrine of the danger of persistent hoarseness so that patients may be seen early, as herein lies the secret of successful arrest of laryngeal carcinoma and at the same time preservation of a functioning larynx.

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LXXI

THE CERVICAL SPINE AND THE GLOBUS SYNDROME

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The complaint of "a lump in the throat" is a common one. The otolaryngologist confronted with this problem remembers that the differential diagnosis insists that he consider postcricoid carcinoma, carcinoma of the pyriform fossa, stricture, Plummer-Vinson syndrome, enlarged styloid process, pharyngeal pouch, pharyngeal paralysis and other conditions. In routine order these various clinical entities are eliminated or verified. There is a group of patients who have had the advantage of such an examination and do not have any of these conditions to explain their complaint. This undiagnosed group is too frequently told that they are neurotic, and for want of a better term, are given the diagnosis of "globus hystericus."

The literature of the last few years has shown a revived interest in the globus syndrome. Some of the contributors have made a sincere effort to present tangible etiology and definitive therapy. Others, apparently mesmerized by the potentialities of the word "hysteria" (defined as a psychoneurotic disorder characterized by violent emotional outbreaks, perversions of sensory and motor functions, and various morbid effect due to autosuggestion) blithely skip over the drab mundane causes and expend their efforts in unravelling a psychogenic background for the complaint. If one is found, and seemingly one can always be found, psychiatric treatment can be expected to produce a cure. The psychiatric approach to this subject is an important one if for no other reason than it is too frequently overused and abused.

This presentation will deal with only one of the tangible causes of the globus syndrome. The complex neuro-anatomy in the cervical spine area can produce symptoms from the diaphragm to the top

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of the head. Osteoarthritis of the cervical spine is one of the causes of the globus syndrome.

The underlying pathology responsible for the peripheral nerve manifestations is a gradual degeneration of the intervertebral disc elements which eliminates the normal cushioning effect and the weight of the head is then imposed on the neurocentral joints. Accompanying inflammation and the production of spurs adds to the insult. These factors produce compression of the cervical nerves. These changes are more common in the lower cervical area at C-5, C-6 and C-7. Naturally the clinical manifestations depend on the exact site and extent of the pathology.

Few patients respond favorably to being told that they are neurotic or hysterical and are resentful of the globus hystericus diagnosis. The patient who has received this diagnosis assumes, often too correctly, that he is being dismissed on the basis that his complaint is a figment of the imagination, that the whole thing is due to a neurosis, and since no specific instructions for either treatment or care were offered, that it is not at all important. Most patients accept this for a time, but the persistence of the symptoms sooner or later drives them to seek further help and advice. In some instances the above routine is repeated several times. Eventually the patient loses confidence in Doctors of Medicine, may truly become neurotic on the basis of frustration, and is in such a state of disorganization as to accept even the most fantastic measures suggested by any charlatan—naturally, for a fee. At least this is a change. Heretofore good money had been spent for no return and in this instance the patient does get something for his money. Whether it is something good or bad, helpful or detrimental, is quite beside the point. It is something.

The average general practitioner and otolaryngologist are adequately trained and equipped to do something more definite, constructive, and beneficial for the patient than to merely suggest that, "it is nothing, just go along your way and forget about it."

History taking is important. The patient should be afforded the opportunity to tell his own story in his own words. The recitation of symptoms, coupled with deviations as to cause and treatment, may be somewhat prolonged but is important to the trained listening ear. It affords an opportunity to determine the mental equipment and attitude of the patient. This supplies a concrete basis

Fig. 1.—The so termed "Poker Spine" showing absence of the normal lordotic curve due to muscle spasm.

for the evaluation of the facts presented, the methods to be employed in determining the cause, the formulation of a plan for treatment, and for anticipating the amount of co-operation that may be expected. As previously stated, the causes of the complaint are many. Only one of the many causes will be considered at this time; namely, the globus complaint due to osteoarthritis of the cervical spine.

It is pathology in this area that produces peripheral nerve symptoms. In order to focus attention on this phase, it is assumed that the hypothetical patient, male or female, approximate age 40 to 50, has had the advantages of a recent general physical check-up and routine examination of the upper respiratory tract including the hypopharynx and larynx. The chief complaint is a sensation of a lump in the throat of eight months' duration. The patient is of a tense type, has seen other doctors, one an otolaryngologist, and seems a bit embarrassed about such a minor complaint; says that maybe it is foolish to even think about the trouble, especially since the family doctor who did an examination just two months ago said everything was fine. Such deviations from normal as were found were stated to be noncontributory and unimportant. A recent radio program and a current lay publication have stressed the importance of paying attention to all minor, chronic conditions. The patient ultimately states that there is a fear of cancer.

The history definitely establishes the absence of dysphagia. There have been no paroxysms of cough, no regurgitation of food and no hemoptosis. There is no family history of cancer, no weight loss nor insomnia, and no history of anemia. In fact, the patient is reported to be in very good physical condition, looks the part, and says that all is well except for the lump in the throat. Questioning brings out these additional facts: The condition is less troublesome at times, is not always present, and days go by when there are no symptoms There is a grating sound in the neck on full rotation of the head to either side, and there is some indication of limitation of motion. There are periods when the muscles of the back of the neck are tight and painful. There are occasional pains, stab-like in character, that involve the greater and lesser occipital nerve distributions. The tight and painful neck muscles are said to be the result of drafts blowing on the neck and are more prevalent during the evening hours. The patient has noted that the lump in the throat is greatly relieved or completely absent for about one hour after eating. Several repetitions of the question relative to pain on swallowing, regurgitation of food, or evidences of a direct blocking when swallowing cannot be accomplished, are interspersed during the review of the history and the examination. In all instances the replies are definitely in the negative.

Examination of the upper respiratory tract reveals no visible evidence of pathology, either local or general, which would have any bearing on the complaint. The examination must be meticulous. While the actual examination of the upper respiratory tract is going on, there is ample opportunity to ask numerous leading questions relative to traumatic injuries to the neck, and to again repeat questions on subjects which may not have been answered too clearly during the history taking period. If the answers to the repeated questions are consistent, there is no reason to pursue this phase any further. Frequently during this period the patient unbends a bit and by the questions asked or the information presented offers the first opportunity the examiner has to evaluate the individual as a real person.

In order to simplify matters we will assume that this hypothetical patient has produced no visible evidences of pathology. The additional history obtained during the period of examination has brought out the fact that there are times when there has been some tingling in the fingers and forearms of one or the other of the upper extremities and there have been periods of occasional pains in the joints of the lower extremities. The patient is usually apologetic about these

Fig. 2.—Slight reversal of the normal lordotic curve. Early arthritic changes showing spurs on the opposing surfaces of the lower cervical bodies.

minor infirmities and invariably has a logical explanation for them. One is often presented interesting therapeutic measures completely devoid of any basic background, pharmacologic, physiologic, anatomic, or even plain common sense, that were the direct cause of the ultimate improvement or temporary disappearance of the associated complaints.

You are now in exactly the same position the previous medical advisers were when they dismissed the patient. One should not feel justified in concluding on the basis that since nothing was found, no tangible etiology was discovered and a comprehensive examination was completed that the patient be told "there is nothing wrong—go along your way and forget about it!"

Experience has shown that the next step may be an important one. The patient is requested to submit to the expenditure of more time and money to obtain x-ray films of the cervical spine and examination of the esophagus (fluoroscopy and films, with and without contrast medium). The radiologist's report frequently states that the examination of the cervical spine shows no evidence of recent or old bony injury; moderate hypertrophic changes are present and involve both the anterior and posterior borders of the fourth, fifth, sixth and seventh cervical vertebrae; there is moderate narrowing of the discs between the fifth and sixth and sixth and seventh and

there are no cervical ribs. Too often the report does not mention the bony changes. These deviations from normal are of utmost importance. Also, it is pleasing to learn that the examination of the esophagus shows no cardiospasm, no defects along the lumen of the esophagus, no diverticula nor any evidence of delay or malfunction in the passage of the bolus of contrast medium.

The ability to read x-ray films with a moderate degree of facility and proficiency is a necessary acquisition. The knowledge obtained from the patient puts you in a most advantageous position to evaluate the roentgenological findings. The radiologist is of invaluable help in pointing out and explaining the minor, yet too often important, key points. Collaboration is essential if the best interests of the patient are to be served efficiently and economically.

The important finding is that there are degenerative arthritic changes in the cervical spine and that there is some diminution in the size of the intervertebral space. The otolaryngologist's realization of the significance of this pathology will make the difference between a relieved and grateful patient and one who is sent away to continue to suffer from the original complaint.

Having obtained this information, disposing of it in a satisfactory manner is another problem. All too frequently the medical co-workers are of little help. The radiologist more or less rapidly tries to efface himself by stating that it is not an uncommon finding, and the degree of change in this particular instance is not great. The internist states that cervical arthritis is a very common condition, may mumble a few words about aspirin, and, although amazed to think that you would suggest arthritis as a causative factor for globus complaint, is wholly unimpressed by your erudite dissertation on the subject. The neurologists and neurosurgeons are of considerable help in explaining some of the complexities of the nerve trunks which arise in this area, the opportunities for referred sensations to and from rather distant areas and for simplifying reflex pathways. The absence of any radiologic or clinical evidence that permits surgical exploration of the area eliminates the necessity for further attention. The average orthopedist, likewise, is of little help. The one or two suggestions relative to fusion of some of the bodies of the vertebrae or a full plaster collar seem a bit too drastic at the moment.

While the diagnostic procedures are in progress, there is no reason why some of the more simple, wholly innocuous but many times

Fig. 3.—Moderately advanced changes showing lipping along the longitudinal ligaments which is more marked in the lower cervical area.

Fig. 4.—Degenerative changes with lipping. There is narrowing of the disc spaces and decrease in size of the intervertebral foramina.

helpful methods, should not be given a trial. Heat in any form, applied to the back of the neck and upper shoulder girdle is usually acceptable and frequently affords great, if not complete, temporary relief. Questioning frequently brings out answers from the patient that substantiate this statement. Whether they sit with their backs to the fire, use an infra-red ray lamp, enjoy an electric pad, obtain relief from soaking in a hot tub, or spend prolonged periods of time with a hot shower directed to the back of their necks, use a hot water bottle, flax seed poultice, or a hot brick or stove lid as a source of heat is wholly incidental. The result is gratifying—at least while the heat is applied. Others obtain similar benefit by the application of strong liniments or other counter-irritants. Further facts to substantiate the beneficial effects of heat may be obtained from a negative approach. Many patients state that they are extremely cautious to avoid exposing their necks to drafts and cold. It is a firmly ingrained practice to protect the neck with scarfs, shawls, or practically any acceptable wearing apparel which will afford protection from the cold. Some of the patients will almost gleefully describe the pleasures of the steam room and a good rub-down. They always omit the cold shower. By and large the response to moist heat is much more effective than the various forms of dry heat. However, there are few of the patients who can routinely afford the luxury of hot packs.

When it is definitely established that there are grounds for pursuing therapeutic measures directed to relieve the peripheral nerve manifestations which have their origin in or about the cervical spine area, intelligent and directed physiotherapy is the first choice. If possible, this should be outlined and supervised by the orthopedist. Haphazard and undirected physiotherapy is seldom satisfactory and when carried to its more complicated phases can be dangerous. I never feel wholly happy while listening to the recounting of the experiences of some of the patients who, either as a result of direction or being somewhat experimentally minded and wishing to conserve on expenses, construct various types of gallows for self-administered neck stretching. Nothing short of a guardian angel could have saved them from hanging themselves with the contraptions they used. In the average community there is at least one doctor who either specializes in treating arthritis or, at least, is willing to suggest and supervise the therapeutic measures. Obviously such a person will care for the patient more efficiently and with less total expense to the patient than one less familiar with this problem.

Radiation therapy has long been used as a therapeutic agent for certain forms of arthritis. When properly administered in small doses over a somewhat protracted period, the results are not only gratifying but persist from shorter periods of several months to longer periods of several years. Doses of 200r once or twice weekly for three or four treatments should not be considered as being small doses. It has been quite surprising to see what very small doses (20r - 30r), twice or even three times weekly for a period of four or five weeks, have accomplished. The total amount of radiation obtained in this way when judged on a basis of time consumed and total dosage permits further similar therapy at some later date. The exact mode of action of this form of therapy is open to considerable discussion. The results obtained in the properly selected cases leave no question in the patient's mind as to the advantages of this type of therapy.

Drugs run the gamut from the popular and generally used salicylates to the later day wonder drugs. Therapy with the vitamins, gold chloride, cortisone, or ACTH has produced spectacular results in some instances.

Therapeutic measures which have proved to be of value are listed in sequence of choice as follows:

1. Physiotherapy (heat, massage, manipulation, traction).
2. Drugs.
3. Radiation therapy.
4. Surgery.
   a. Esophagoscopy. The trauma associated with the procedure frequently results in regression or elimination of symptoms.
   b. Fixation—fusion—removal of spurs.

The various names by which the cervical vertebra have been known indicates that as a result of appearance, position or function, certain of them have been set apart from the rest of the members of the vertebral column and even from others of the cervical group for special attention. C-1 and C-2 have long been known respectively as the atlas and axis or epistropheus. These two have been grouped under the heading of rotation vertebrae. The remaining members of the column have been described as flexion vertebrae. C-7 is also known as the vertebra prominens because its spinous process projects beyond all others.

Fig. 5.—Advanced changes showing lipping, bridging and some narrowing of the disc spaces.

C-1 and C-2 differ from the others in this group. The outstanding features are that C-1 has no body and C-2 has a large odontoid process which rises perpendicularly from the body. These anatomical differences offer increased opportunities for nerve injury by trauma as well as insult resulting from inflammation and encroaching calcium deposits. The nerve roots of NC-1 and NC-2 emerge behind the lateral articular masses and are not protected posteriorly by the pedicles and facets which complete the root canal elsewhere in the vertbral column.

A consideration of the mechanics of the cervical spine shows that with the exception of C-1, there are five joints between the vertebrae: The articular facets, the intervertebral disc and the two small synovial joints on the posterior lateral margins. These last two are frequently referred to as the lateral intervertebral joints and some authors consider these to be the only true joints. The ligaments and the deep fascia divide the spaces into many compartments.

There is limited motion between the atlas and the occipital bone. This fact plus the knowledge that the first cervical nerve is usually devoid of a sensory component readily explains why this nerve root is seldom accused of playing a part in the production of unpleasant symptoms.

There is marked mobility of the joint between C-1 and C-2. Of the eight cervical nerves, C-2 is the most liable to suffer from trauma. The posterior ramus of NC-2 which makes up the bulk of the greater occipital nerve emerges between bony surfaces. The anterior ramus is liable to unusual stress under normal conditions. There are tremendous excursions of the articular facets of C-1 and C-2 in ordinary rotary motions of the head. NC-1 and NC-2 send branches to the tenth and eleventh cranial nerves by way of the cervical plexus.

Roentgenologic evidence of cervical spondylosis is most commonly seen in the areas subjected to maximum stress and strain resulting from flexion and extension. C-4, C-5 and C-6 are the areas which usually show the degenerative changes. Changes in the lateral intervertebral joints or shortening or encroachment on the neuroforamena by bone fringes, osteophytes or web-like calcifications satisfy the criteria for diagnosis. It should be accepted that the visible changes seldom represent more than fifty per cent of the actual change. The roentgenograms do not show the accompanying soft tissue changes.

The gradual degeneration of the intervertebral disc elements eliminates the normal cushioning effect. The neuro-central joints must then support the weight of the head and this produces opportunities for compression of the cervical nerves. The known opportunities for intercommunication and referral of afferent and efferent impulses between the cervical nerves and the cranial nerves V, IX, X and XI are almost legion. The literature on this subject is extensive but far from complete.

Further complex neuro-anatomy of the area has been deliberately omitted. Any simplified abstracted form could not be instructive or interesting. A point of progress has been made if this presentation stimulates a sincere desire to review the neuromuscular anatomy of this area. Cunningham, Grey, Pierosol, Toldt and other standard texts contain a surprising amount of information which is both informative and really pleasant reading. If a mind is sufficiently plastic, is still inquisitive and is willing to accept a challenge, it is offered in the form of a suggestion to review the neuro-anatomy of this area and consider the practical applications. The results of such a review will afford many advantages in handling most of these problems.

Fig. 6.—Marked osteoarthritic changes. In addition to some difficulty with swallowing there was occasional difficulty with respiration and a muffled somewhat slurring speech.

It is only in the last few years that osteoarthritis of the cervical spine, less well known as cervical spondylosis, has been given any consideration as being the cause of serious neurological disorders. It is seldom mentioned as a cause of dysphagia. The fact that cervical arthritis is never listed as the immediate cause of death explains why the routine postmortem examinations do not pay attention to this area. The available histological examination of tissues from this area is very scanty.

It is realized that cervical spondylosis is only one of the many causes of the globus syndrome. The management and treatment of the cervical spine pathology is not fundamentally within the field of otolaryngology. It is fitting that the patient should consult the otolaryngologist in regard to discomfort in and about the throat. It should be satisfying to both the examiner and the patient to have eliminated the immediate fear of cancer, establish a diagnosis and offer concrete suggestions for treatment. The responsibility of the otolaryngologist does not cease with the establishment of a diagnosis. The patient should be directed to the proper sources for intelligent treatment and should be requested to report for follow-up examinations. These subsequent visits should be based on the patient's progress.

The figures demonstrate varying degrees and types of pathology. All of the subjects had the one common complaint of "a lump in the throat."

CONCLUSIONS

1. Pathology of the cervical spine is presented as one of the tangible causes of the globus syndrome.

2. The complex neuroanatomy of the cervical spine area readily explains peripheral nerve manifestations in regions from the diaphragm to the top of the head.

3. A tentative diagnosis of osteoarthritis of the cervical spine may be made from the clinical history. Roentgenograms will confirm or negate the tentative diagnosis.

4. The relief of symptoms following simple therapeutic measures will substantiate the diagnosis.

5. Cervical arthritis should be ruled out as a tangible cause for the complaint of "a lump in the throat" before the patient is advised to forget about the discomfort or is sent for psychiatric evaluation and treatment.

490 Post St.

LXXII

FUNCTIONAL DISTURBANCES OF THE UPPER SWALLOWING MECHANISM

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The reason for bringing this subject up for discussion is that it constitutes one of the most common problems to be met with in the practice of the specialty.

The term "functional" is defined in the medical dictionary as implying some alteration in the action of an organ or a mechanism in contrast to a change in its structure. The discussion of this type of disturbance of the swallowing mechanism may include three main groups: Those due to changes in the action of the musculature, those due to alterations in the innervation and those caused by disturbances of psychic control. Of the three stages of swallowing[1] the second stage in which the food passes through the pharynx into the upper end of the esophagus is the most complicated.

In the pharynx the airway and the food passage have a common pathway. This necessitates the presence of sphincter mechanisms to protect the airway from the entrance of food, both at the nasopharyngeal and laryngeal openings as well as a sphincter at the junction of the pharynx and esophagus to isolate or protect the airway from contents of the esophagus and prevent entrance of air[1] into the esophagus during inspiratory effort.

The most important muscles which take part in the propulsion of food through the pharynx are the muscles of the soft palate, the constrictors and elevators of the pharynx, and the muscles which close the upper aperture of the larynx.

The action of part of this system of muscles can be observed on direct examination, for example the tongue, palate, and the constrictors of the laryngeal aperture. Also some of the symptoms caused

From the Division of Otolaryngology of the University of Chicago.

The x-ray pictures have been made by the Department of Roentgenology at the University of Chicago. Some of the illustrations have been used in *X-ray Examination of the Stomach* by Frederic E. Templeton, University of Chicago Press, 1944.

Fig. 1.—Normal pharynx after swallowing barium. a. Pharynx during the resting stage after swallowing barium. b. Pharynx during the Valsalva test. The cricopharyngeus muscle is outlined by this maneuver.

by weakness or failure of these muscles to function are readily recognized. The tongue may be unable to propel the bolus into the pharynx. Defective action of the palate may cause an escape of fluid or food into the nasopharynx and nose, interference with articulation of speech and rhinolalia operta. When the upper aperture of the larynx fails to close tightly there may be an escape of fluids into the airway with resulting cough and the possibility of complications from aspiration. Also if the glottis does not close adequately there may be air loss during speech and interference with such acts as the hiccough and laughing.

The action of the constrictor muscles of the pharynx and of the cricopharyngeal sphincter can be demonstrated best by fluoroscopic examination and x-ray pictures during and after swallowing a thick barium mixture. Spot films are used to record certain phases of the action. Special positions and manipulations may be used to get

increased information. The Valsalva maneuver[2,3] just after swallowing with the patient in the straight lateral position as well as in the anterior-posterior position is particularly helpful in demonstrating any weakness or asymmetry of the pharyngeal constrictors and any failure of the sphincteric action of the cricopharyngeus muscle. (Fig. 1)

DISTURBANCES OF MUSCULAR ACTIVITY

Those diseases which may cause changes in the action of the musculature without obvious localized or gross changes in structure include the following: Myasthenia gravis, diffuse generalized scleroderma, dermatomyositis and myasthenia associated with some systemic diseases; i.e., tuberculosis, thyrotoxicosis and myasthenia associated with advanced senility.

Advanced myasthenia gravis may so impair muscular action as to prevent adequate closure of the glottis and the upper aperture of the larynx causing a tendency for fluids to enter the airway. There may be impairment of the ability of the constrictors to move the bolus along and to empty the pharynx (Fig. 2). On x-ray examination the barium tends to remain in the valleculae[4] in this as in most disturbances which decrease the muscular activity of the constrictors.

In some cases of diffuse scleroderma and dermatomyositis the diffuse changes in the connective tissue and muscle may cause a similar functional disturbance. The laryngeal symptoms may be more prominent. Not only may liquids tend to enter the airway but the patient may have loss of air during speech and be unable to laugh or hiccough.

In advanced senility there may be asthenia, impairment of the gag reflex and defective closure of the upper aperture of the larynx (Fig. 3). Food or barium may stick in the valleculae and pyriform sinuses and liquids tend to escape into the larynx. Solid foods may be swallowed with less difficulty.

A similar type of generalized asthenic state has been observed in thyrotoxicosis and in tuberculosis without evidence of a local lesion in the pharynx.

The inability of the pharyngeal musculature to cleanse itself of the barium despite repeated swallowing may be evident on fluoro-

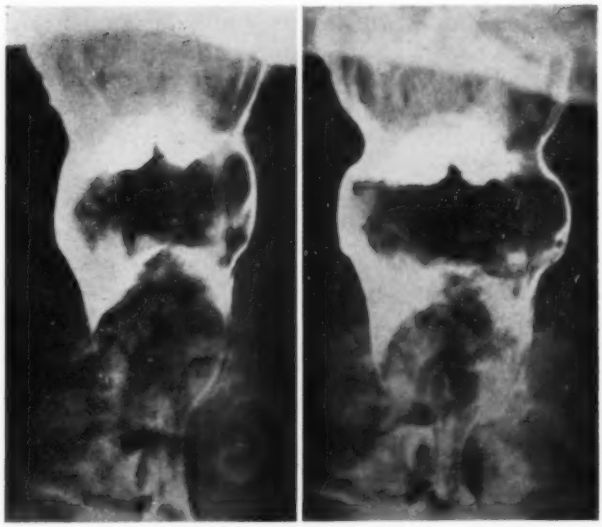

Fig. 2.—Generalized scleroderma. a. Resting stage after swallowing barium. Considerable barium remains in the valleculae and pyriform fossae because of impaired muscular contraction. b. During the Valsalva test.

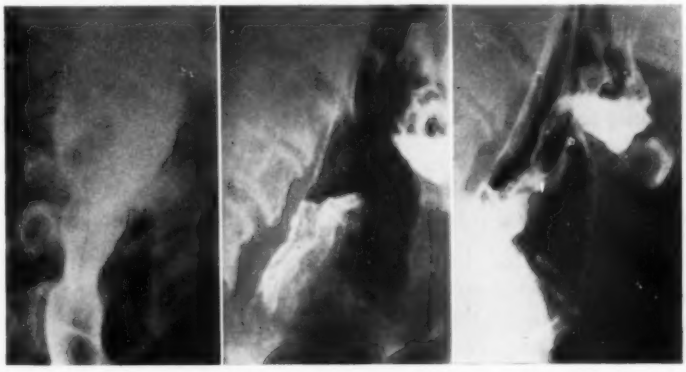

Fig. 3.—Disturbance of swallowing in the aged. a. Solid food and thick barium passes with no difficulty. b. Barium and food are retained in the vallecula due to generalized asthenia. Exostoses are seen on the vertebra. c. Some barium is seen in the larynx. Fluids tend to enter the airway on swallowing.

scopy. The cricopharyngeus muscle tends to retain its effectiveness as a sphincter and to prevent the escape of barium from esophagus to pharynx during the Valsalva maneuver. The interpretation has been made[5] that the failure of the pharynx to empty represents achalasia or a failure of the cricopharyngeus sphincter to open, however, it would seem that the primary disturbance is of the pharyngeal musculature as a whole.

DISTURBANCES OF INNERVATION

These include both motor and sensory disturbances.

The motor nerve supply to the pharyngeal constrictors and the muscles of the glottis is the vagus. The tensors of the palate are supplied by the V nerve while the levators are said to be innervated by the vagus through the pharyngeal plexus.[1] The sensory supply to the pharynx is from the IX and X nerves.

Motor disturbances usually cause paresis but in some instances may cause spasm. The most common causes of paresis are:

1. Peripheral neuritis: This has commonly been observed after diphtheria and may follow other acute infections.

2. Jugular foramen syndrome: This includes a number of syndromes in which there are varied combinations of paralyses of palate, pharynx, larynx, tongue, neck and shoulder (Tapia, Avellis, Schmidt, Vernet, Jackson, Collet-Sicard) due to lesions in the region of the jugular foramen.

3. Lesions affecting the nerves as they leave the bulb and enter the jugular foramen, i.e. meningo-encephalitis, tumors, fractures, etc.

4. Intracranial lesions: Hemorrhage and embolism affecting the bulb, tumors pressing on the bulb, tabes dorsalis, poliomyelitis, disseminated sclerosis, syringomyelia and amyotrophic lateral sclerosis.

The diagnosis of disturbances affecting the innervation of the pharyngeal muscles is made partly upon the history, the symptoms and signs referable to the pharynx and the associated indications of neurological disease. The fluoroscopic examination and x-ray photographs are useful diagnostic aids in demonstrating the disturbances of muscular action of the pharynx.

In the presence of a severe paresis as in a bulbar palsy the examination with barium should be made with caution because of danger

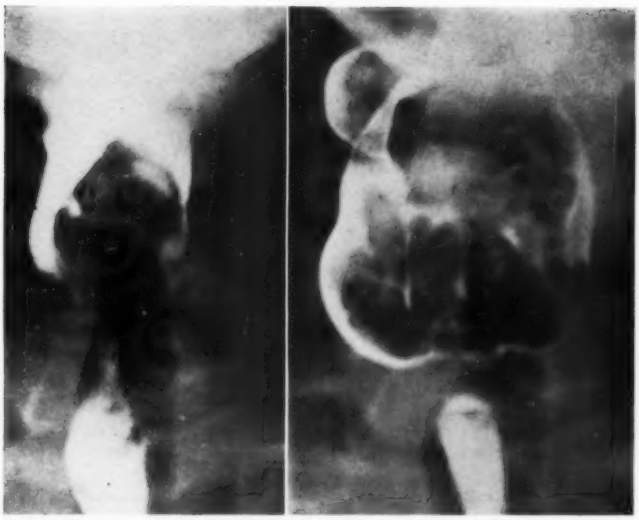

Fig. 4.—Unilateral paresis of pharyngeal muscles due to intracranial lesion. a. Barium remains in the right pyriform sinus after the bolus has passed. b. During the Valsalva test the right side of the pharynx balloons out more than the left.

of aspiration. Thick barium is less likely to be aspirated than a thin mixture. A bilateral weakness of the neuromuscular mechanism may cause a symmetrical delay in the emptying of barium from the vallecular and from the pyriform sinuses. A unilateral weakness may show a tendency for barium to remain in the pyriform fossa of the affected side and for that side to bulge outward to a greater extent on the Valsalva test (Fig. 4).

The cricopharyngeal sphincter tends to retain its function in unilateral pharyngeal paralysis (Fig. 5). In bilateral paralysis such as may occur in poliomyelitis or tabes (Fig. 6) the sphincter may lose some of its efficiency. Esophageal contents may tend to leak back into the pharynx on performing the Valsalva maneuver in the recumbent position.

The examination of the pharynx during the Valsalva maneuver therefore becomes a useful method to demonstrate a failure of the sphincteric action of the cricopharyngeus muscle.

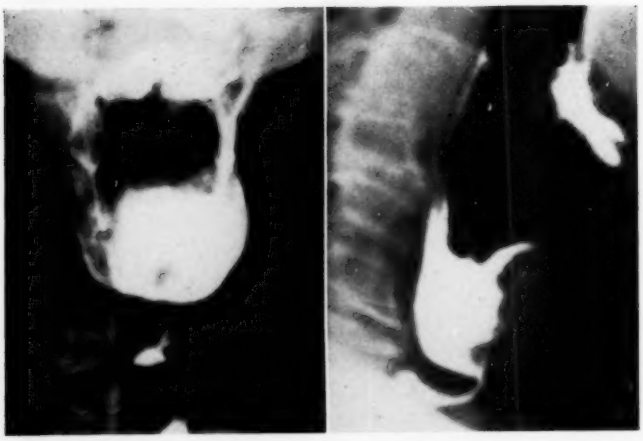

Fig. 5.—Unilateral pharyngeal palsy due to vascular accident. a. After swallowing barium is retained in the valleculae and in the pyriform fossa on the paralyzed side. b. During the Valsalva test the cricopharyngeus muscle is demonstrated and appears to retain its function as a sphincter.

Spasm. A tonic spasm of the pharyngeal musculature may occur in disorders of the central nervous system such as tetanus or brain injuries as well as in acute local infections. A tonic spasm probably also occurs in some psychogenic disorders (cricopharyngeal spasm, globus hystericus). The demonstration of such a spasm by means of x-rays has not yet been a practical possibility.

A disturbance which may have some relation to cricopharyngeal spasm is the development of a pulsion diverticulum. In the early stage the diverticulum may be observed to project backward with part of the sphincter muscle above but most of it below (Fig. 7). The pouch develops at the weakest point, where oblique and transverse fibres separate probably as a result of disturbed co-ordination of the sphincter.

The occurrence of unexplained pain in the region of the cricoid cartilage which is relieved by swallowing may suggest spasm of the cricopharyngeus.

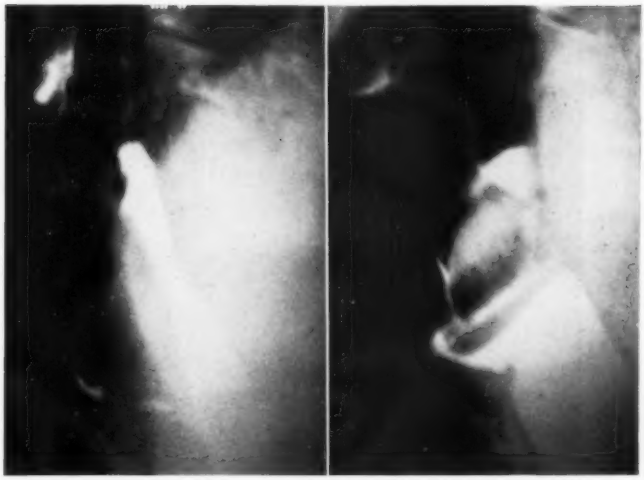

Fig. 6.—Impaired action of the cricopharyngeal sphincter in tabes dorsalis. a. Barium passes freely from the pharynx into the esophagus. Some has escaped into the larynx indicating either sensory loss or impaired muscular action at the upper aperture of the larynx. b. During the Valsalva test barium in the esophagus was forced upward against the cricopharyngeal sphincter. Impaired action of the sphincter allowed barium to escape into the pharynx and some to enter the larynx.

A chronic rhythmic contraction affecting the pharyngeal muscles along with the palate (palatal nystagmus) may occur. Visible rhythmic movements of the upper part of the neck may sometimes be detected. A clicking sound coming from the eustachian tube is likely to be the major complaint. The cause may be obscure but central nervous system disease is to be suspected.

DISTURBANCES OF PSYCHIC ORIGIN

In contrast to muscular and neural lesions the frequency of a psychic disturbance of the swallowing mechanism is high. Some of the complaints referable to the pharynx which may occur on this basis are pain, irritation, burning, dryness, choking or the sensation of a lump in the throat.

These disturbances are psychosomatic in the strict sense in that alterations in secretory activity as well as muscular contraction are

Fig. 7.—Early pharyngoesophageal diverticulum. a. The small diverticulum remains filled with barium after swallowing. b. During the Valsalva test the relation of the diverticulum to the contracting cricopharyngeal muscle is demonstrated.

probably a part of the process and produce the sensory stimuli. In occasional instances it has been found that a psychic disturbance occurred secondarily to an organic disease. Severe anxiety or depression has sometimes been the most prominent manifestation in early malignancy.[6]

Dysphagia of psychogenic origin varies widely in severity. In milder forms there may be the complaint of a lump in the throat which does not interfere with swallowing food. Fluids do not enter the airway. In these less severe types the common cause is the fear of cancer or other serious disease. A careful examination of the

swallowing mchanism is an essential part of the procedure to help rule out organic disease (i.e., early tumor, stricture, Paterson-Kelly or Plummer Vinson's syndrome, etc.).

In cases of psychogenic origin the x-ray examination is usually reported as showing no abnormality in the swallowing mechanism. While spasm or increased tonic contraction of the cricopharyngeus probably occurs it cannot be distinguished from the normal by the technique now in use.

The endoscopic examination may also be essential inasmuch as early malignant disease may be present without clear x-ray evidence.[7]

In practice it may be preferable to delay further examination procedures in some cases until the effect of the initial investigation and reassurance has been observed, but if relief is not produced promptly the endoscopic examination should be made.

In the more severe psychogenic types there may be inability or failure to swallow solid food. The bolus may not be passed into the pharynx from the mouth or it may be rejected. The action of the esophageal musculature as shown by x-ray may not be altered. These severe disturbances of swallowing are seldom due to a simple fear of malignancy or serious disease. They are commonly found in the more severe types of mental illness such as neurosis, hysteria or sometimes a psychosis. The complete examination is necessary in order to exclude a primary organic disease but reassurance may be of no help. The treatment in such cases requires the services of a psychiatrist.

In all cases in which the presence of a psychogenic dysphagia is suspected or indicated it is advisable to avoid all forms of local therapy. Treatment must be directed to the basic psychic disturbance if lasting results are to be obtained.

SUMMARY

Functional disturbances of the upper swallowing mechanism refer to alterations in the action of the mechanism as contrasted to any change in structure.

Functional disturbances may be divided into three groups—those affecting musculature, innervation and psychic control.

Direct observation of the action of the swallowing mechanism is not feasible but useful objective evidence can be obtained by fluoroscopy and x-ray films.

Weakness or asymmetry of action of the constrictor muscles and of the cricopharyngeus commonly seen in disturbances of musculature and innervation can be confirmed by x-ray examination. Evidence of increased tone or spasm of these muscles has not been afforded by present techniques.

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LXXIII

CARCINOMA OF THE MAXILLARY SINUS IN A YOUTH OF TWENTY

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Squamous cell carcinoma of the paranasal sinuses is said to occur infrequently in the first three decades of life. Having encountered such a malignancy in the maxillary sinus of a youth of twenty years of age, we considered it worth bringing to your attention.

REPORT OF A CASE

G. J., aged 20, white, was referred to Dr. Franklin, of Ramsey, Illinois on December 1, 1950.

The patient stated that about four weeks previously he had noticed a numbness over the left side of his face and cheek from midline to the zygoma which gradually spread about his left eye. This was followed one week later by a moderate amount of painless swelling of his left cheek which slowly extended upward, until one week ago when his lower and upper lids became markedly puffy, lacrimation and purulent discharge from between the closed lids and the left side of his nose became marked, and his left eye seemed to be more prominent than the right, though not painful. The purulent discharge from the left side of his nose, though copious recently, had never been sanguinous, nor had epistaxis occurred. There had been no trismus. The hard palate did not show evidence of involvement. The sight was good in the right eye though blurred in the left. During this time his physician treated him with injections of penicillin, despite which his condition became progressively worse. He had had a fluctuating low-grade fever but no chills. He was not subject to nasal infections and had always been in good health.

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Fig. 1.—December 7th. Note proptosis of the left eye, edema of lids and chemosis of conjunctiva.

Fig. 2.—December 7th. Lateral view further accentuates features in Figure 1, and demonstrates absence of enlargement of cervical glands.

Examination. This revealed a well-nourished white male, mentally responsive and co-operative. His general physical, functional and neurological condition evidenced no abnormalities.

The lids of his left eye were extremely edematous, slightly purpuric and there was marked chemosis of both. The globe was moderately proptosed downward and outward and partially fixed. It was not painful to palpation. There was numbness over the left cheek and no tenderness on deep pressure over the antrum or frontal sinus. He was able to distinguish individual fingers at a few inches. The right eye was apparently normal but somewhat injected. The mucosa of the left side of his nose was intensely red, the turbinates were engorged, there was a moderate amount of pus on the floor and in the left middle meatus in which the mucosa was edematous, slightly polypoid and bled easily when touched. No growth was seen. The mucosa of the right side of his nose was generally reddened and there was some pus on the floor but none seen in the meatuses. His throat and ears did not show any significant pathological changes. There were no enlarged or tender glands about the head or neck (Figs. 1 and 2).

He was hospitalized immediately at Firmin Desloge Hospital in St. Louis. On admission to the hospital on December 1, 1950,

his temperature was normal, white blood count was 10,000, the differential normal, and the red blood count 5,500,000.

The x-ray of the paranasal sinuses on December 1, 1950, showed a moderate density of the left ethmoids, anterior and posterior, and a marked density of the left maxillary sinus. Both frontal and sphenoid sinuses as well as the right ethmoids and maxillary were clear.

Our tentative diagnosis was: 1) Acute purulent hyperplastic maxillary and ethmoidal sinusitis, left. 2) Retrobulbar edema, cellulitis, or abscess, left.

On December 2, 1950, under intratracheal ether anesthesia, the conventional Caldwell-Luc approach to the left antrum was made. The anterior bony wall was not bulging, but was thin and somewhat soft. The sinus was entered through the canine fossa and found to be filled with reddish, edematous mucosa, which bled freely when curretted and contained a small amount of pus. The mucosa was friable to the extent of not being cohesive and not adherent to the sinus walls. After the sinus had been curretted cleanly, there was seen to be a linear dehiscence about 1 x 2 cm in the medial wall into the inferior meatus. The bone of the superior portion of the medial wall was intact. Another dehiscence of about the same extent was also observed in the medial third of the orbital plate, and tissue similar in appearance to that found in the sinus was seen within the orbital fossa, except that it was not friable and did not bleed as actively. The fistula through the orbital plate was enlarged to obtain better drainage; no pus was obtained. The operation was completed by removing more of the nasoantral wall of the inferior meatus and a vaseline gauze drain placed in the opening.

The pathologist's report was: *Gross:* "Several pieces of reddish-tan tissue, the surfaces covered with a thick smooth, whitish membrane. There were also several fragments of bone." *Microscopic:* "Reveals large anaplastic basophilic pyramidal epithelial cells arranged in irregular tracts and clusters and surrounded by dense connective tissue: mitotic figures numerous. A few bony trabeculae between which clusters of anaplastic cells are seen." *Diagnosis:* Squamous cell carcinoma, poorly differentiated. (Figs. 3 and 4)

The pathologist expressed the opinion that this type of tumor should be radio-sensitive, which was concurred in by the radiologist, who advised irradiation with deep x-ray. Accordingly the patient

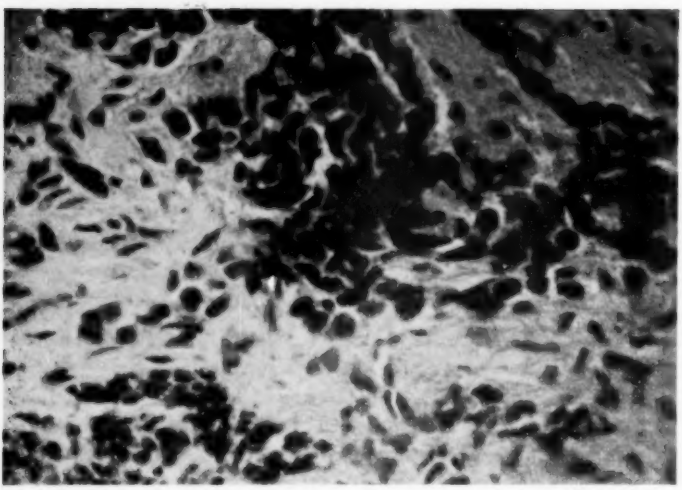

Fig. 3.—*Before irradiation*: High power photomicrograph showing highly anaplastic epithelial cells infiltrating connective tissue spaces. Note marked nuclear hyperchromatism and relative absence of cytoplasm.

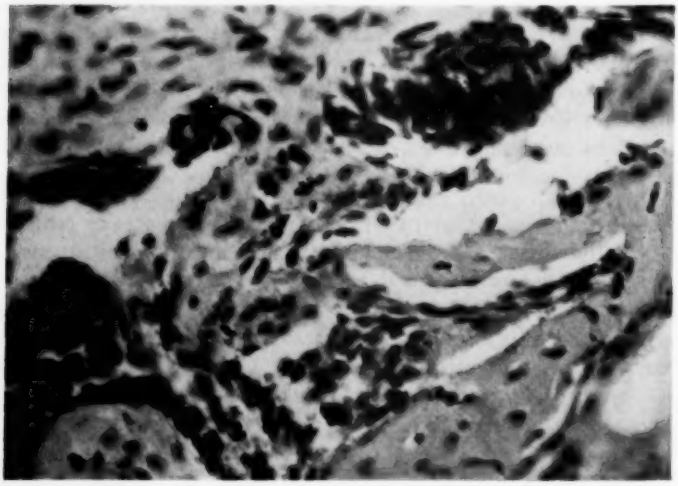

Fig. 4.—In this field tumor cells are seen invading bone of naso-antral wall. Small size and ragged edges of spicules indicate secondary osteolysis.

received daily treatments of 200 R. over a 5 x 7 area of the left side of his face, from December 9, 1950, to January 26, 1951, thirty-nine exposures: a total of 7800 R.

Within a few days the swelling about the left side of the face and eyelids began to subside, and in two weeks the proptosis had almost completely disappeared. About six weeks after x-ray treatments had been started, there was some improvement in the ocular movements and perception, though the eye was still injected, and there was some increase in sensation over the left cheek. In the meantime the intranasal condition had improved and the airway was clearer, but a small fragment of polypoid tissue removed from the left middle meatus with suction revealed a few malignant cells deep in the tissue (Fig. 5). However, at about this time there was noticed a large firm fixed mass in the left side of the neck just inferior to the angle of the mandible (Fig. 6). This mass was not tender or fluctuating. There was no trismus. Because of the status of the entire malignant picture it was decided that surgery of the neck glands was inadvisable. Deep x-ray therapy to the neck glands was inaugurated, and the patient received 1800 R. over a period of nine days without perceptible beneficial effect upon the glands, in fact, all anterior cervical glands and a few in the posterior chain were then enlarged and the original glands were markedly increased in size. Shortly thereafter he developed severe intractable pain in the left shoulder and chest, and pain and tenderness over the right sacroiliac area. X-ray of these areas did not show tumor formation. Realizing that metastases were becoming generalized, x-ray therapy of the neck was discontinued.

Since the eventual outcome was inevitable the patient was discharged on March 5, 1951, and returned to his home where he died within two months with symptoms of intracranial involvement and paralysis of the lower half of his body. The autopsy was not done.

REVIEW OF LITERATURE

While no attempt has been made to review the entire literature on malignancies of the paranasal sinuses, since our presentation is not that extensive, it is pertinent to mention that about 0.2 per cent of cancers in humans, or as high as 2 per cent in cancer hospitals, occur in the paranasal sinuses, according to Wilkins.[1]

In reviewing 295 cases of malignancies of the upper jaws and antrum, New[2] reported an incidence of squamous cell carcinoma of

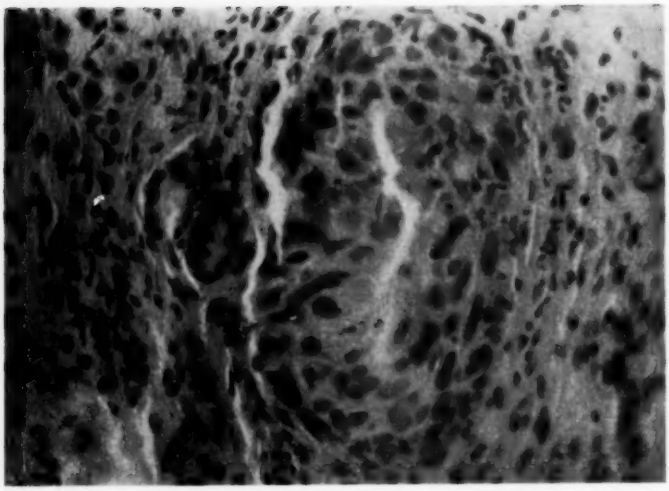

Fig. 5.—*After irradiation*: Small polyp from left middle meatus. A nest of atypical epithelium is seen surrounded by connective tissue with many inflammatory cells. Although the pleomorphism and lack of polarity point to to the true nature of these cells, they are obviously less anaplastic than those seen in the previous photomicrographs.

66 per cent in the maxillary sinuses. Öhngren[3] reported 187 cases with an incidence of 56.6 per cent in the maxillary sinuses. Ringertz[4] reported 218 cases, with an incidence of 86.7 per cent carcinomas in the maxillary sinuses.

In reporting 168 cases of malignancies of the paranasal sinuses, New[5] has stated the numerical occurrence of the carcinomas, the sarcomas and the mixed malignancies in decades, but not by the sinuses or by each type of malignancy. Robinson[6] in reporting 60 cases has done likewise, and Houser[7] 21 cases of the antrum alone, 19 being carcinoma. Öhngren[3] has reported 149 cases of all types in decades, but he has been more specific in his case reports. He[3] and Ringertz[4] gave a detailed history of the youngest patient on whom such an accurate case history could be found. This was a woman aged 25 whose condition was diagnosed as squamous cell carcinoma, with hornification, of the maxillary and ethmoid sinuses and the orbit, in 1930.

The description of the physical appearance of this patient was similar to that of my patient, except that the right nasal cavity was filled with soft readily bleeding tumors, the ethmoidal area was necrotic and bulged into the right nasal cavity, and the mandibular angle glands were enlarged. The onset of the intranasal symptoms occurred five months before the patient reported at the clinic. The patient was treated with electrosurgery, radium and irradiation. She died with pulmonary metastases one year and four months after commencing treatment.

TABLE 1.

REPORTED AGE INCIDENCE.

Age	New[5]	Robinson[6]	Houser[7]	Ringertz[4]	Öhngren[3]	Totals
1-10 Yrs.	3	2	0	0	0	5
10-20 Yrs.	7	3	1	0	1	12
20-30 Yrs.	4	6	0	2	5	17
Total	168	60	21	218	149	616
Types	Ca. Sarc. Mixed	Ca. Sarc. Mixed	Ca. Sarc. Antrum	Carcinoma	Ca. Sarc. Mixed	

While Heatly[8] states that tumors in children under 15 are "not uncommon," the foregoing figures do not bear out this statement. However, since these reports include malignant tumors of all types, with the exception of Ringertz' and Öhngren's cases, we have no way of knowing how many and at which ages squamous cell carcinomas occurred.

SUMMARY

This report is submitted for your consideration with two purposes in mind, first, to bring out the fact that squamous cell carcinoma may occur in the paranasal sinuses of a younger person, and second, to bring forth comments relating to the treatment of such a condition.

Our original presumptive opinion was that the patient was suffering from an acute suppurative hyperplastic left maxillary and ethmoidal sinusitis, and a left retro-orbital cellulitis or abscess. This conclusion was arrived at because of the age of the patient and the rapidity of the onset and development of his symptoms. Because

Fig. 6. February 21st. Seven weeks after start and three weeks after cessation of x-ray therapy. Note absence of proptosis left eye and very little edema of upper lid. Prominence of left side of neck caused by enlargement of anterior cervical glands.

the first symptom was a numbness over the left side of the face, indicating pressure on the infraorbital nerve, we presumed that the left maxillary sinus was the site of the original infection, and that it had extended into the orbit and thence to the ethmoidal sinuses. This extent of involvement was corroborated by the report of the x-ray examination of the sinuses. Immediate surgical measures were taken to relieve the pressure within the maxillary sinus. The findings at operation suggested a subacute purulent papillary sinusitis. However, the pathologist's report of squamous cell carcinoma in the tissue removed caused great concern.

Irradiation was decided upon as the optimum method of treatment because the lesion was so extensive, involving the entire left ethmoidal labyrinth and the retrobulbar orbital contents, that it did not seem possible to encompass it satisfactorily by surgery, especially since the microscopic section showed involvement of the bony walls of the maxillary sinus, implying a similar involvement of the walls of the ethmoidal sinuses, and the possibility of deeper extension into the cranial cavity. Likewise, the pathologist and radiologist agreed that the tumor should be radio sensitive, judging from its type of

architecture. Although Ringertz[4] has stated that "it is difficult to determine the radio sensitivity of the various architectural types, in carcinoma of the nasal and paranasal sinuses, since the deep position of the tumors and the preponderance of operative treatment greatly limits the possibilities of grading the sensitivities."

The ophthalmologist and the radiologist were consulted regarding the enucleation of the left eye and they agreed that no advantage would be gained by so doing since no gross inequality between the optic foramina could be seen by x-ray, and there was some hope of saving the eye. The radiologist felt that the eye would not materially interfere with the penetration of the therapeutic x-rays to the retro-orbital and ethmoidal areas.

The progress towards recovery seemed to be favorable for the first six weeks after x-ray treatments were started, until the enlarged cervical glands were noted at the left mandibular angle, indicating the onset of metastasis. It was then but a few weeks before generalized metastases became established and death ensued.

The impression is prevalent that carcinoma of the maxillary sinus metastasizes late. Öhngren expresses the opinion that this impression is false, since the patient rarely comes to be examined early in the disease and also, that the first palpable gland is usually located in the deep jugular chain, secondary to the lateral retropharyngeal lymph glands which must have been involved at an earlier date. He very clearly traces the lymphatics from their "rich network in the mucosa of the antrum of Highmore with their numerous anastamoses." He goes on to say, "submucously we find larger stems running in a direction towards the opening of the maxillary antrum, diminishing in number but increasing in calibre. They leave the antrum here and before long join the lymph-vessels of the middle meatus, these vessels subsequently passing backward towards the choanae running together into a plexus in the lateral part of this wall, immediately anterior and superior to the eustachian orifice. From this plexus a few large lymph channels are given off backwards above as well as below the eustachian cushion towards the posterior pharyngeal wall. Here they pick up finer branches from the nasopharynx before entering the retropharyngeal space. The lateral retropharyngeal lymph nodes receive most of these lymph vessels, only small branches pass directly upwards emptying into the medial jugular glands located just below the base of the skull. From the lateral

retropharyngeal lymph glands the lymph is conducted in stems of larger calibre which empty into a node belonging to the deep jugular chain and usually located at the level of the carotid bifurcation."

Ringertz[4] states that it is frequently impossible to determine whether a carcinoma is primary in the maxillary sinus or in the ethmoid when both are involved, because the majority of such patients are not seen early enough. Our patient was seen within one month of the onset of his symptoms. The first symptom, numbness of the left cheek, antedated by three weeks the orbital symptoms indicative of extension into the retro-orbital space from the antrum and probably the ethmoids, causing the proptosis and fixation of the eye. The microscopic picture of the invasion and destruction of the bone by the cancer cells illustrates the 'modus operandi' of these cells in their process of direct extension.

CONCLUSIONS

1) The report of a squamous cell carcinoma of the maxillary sinus and probably of the ethmoids, in a youth of twenty years of age is presented.

2) Treatment consisted of a radical exentration of the contents of the antrum followed by deep x-ray therapy of 7800 'R' within a period of seven weeks, and later 1800 'R' to the glands of the neck.

3) The patient died of generalized metastases six months after the symptoms were first noted and five months after treatment was started.

4) The sequence of symptoms suggest that this tumor occurred primarily in the maxillary sinus.

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I wish to express my appreciation to the following for their valuable assistance: Photograph, Mr. P. A. Conrath; photomicrographs and kodachromes, Dr. Thomas Tombridge, Department of Pathology. Pathological diagnosis, Dr. Henry Pinkerton, Director of the Department of Pathology.

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LXXIV

A MOTOR PATTERN IN THE NUCLEUS AMBIGUUS. ITS CLINICAL SIGNIFICANCE

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With the beginning of knowledge of laryngeal physiology the imaginations of laryngologists were captured by the almost unlimited possibilities of motor function. The complex and intricate mechanisms of the larynx both as regards the action of the sphincters and the innumerable relationships the cords may assume in harmonious action with each other have long suggested the logical practicality of a pattern for the cells of origin for the superior laryngeal and recurrent laryngeal nerves. We now hold the conviction established by evidence that such a pattern does exist in the nucleus ambiguus of the monkey.

The nucleus ambiguus is a motor nucleus extending from the upper border of the medulla and the region of the striae medullaris caudalward to the level of the motor decussation. It is generally recognized to be the origin of motor fibers to the striated muscles of the pharynx, larynx and the esophagus. These muscles are derived phylogenetically from the gill arch musculature. They are supplied by the glossopharyngeal, vagus and bulbar accessory nerves (branchiomeric special visceral efferent nerves). The pattern of cells of origin in the nucleus ambiguus for the larynx is particularly difficult to determine because the nucleus is long and composed of cells arranged in a complex manner. It has been described for carnivores (cat) by Szentagothi.[1] He placed small electrolytic lesions in the nucleus which he later studied. After a suitable interval he also observed the degeneration of the motor end plates in various laryngeal muscles. Thus he was able to establish a definite pattern with the following arrangement: 1) the cricothyroideus muscle in the rostral part of the nucleus and 4 mm rostral to the obex level. The remaining muscles were innervated in the following order behind the crico-

Fig. 1.—Normal motor neurones in the nucleus ambiguus of the monkey. Magnification approximately 90 x.

thyroideus and all rostral to the obex; 2) cricoarytenoideus dorsalis 2 - 4 mm in front of the level of the obex; 3) thyroarytenoid 2 - 3 mm rostral to the obex; 4) lateral cricoarytenoid 1 - 2 mm and 5) arytenoideus 1 mm in front of the obex.

In a series of experiments upon monkeys, a comparable pattern was obtained except that the innervation of the cricothyroideus was found to be slightly caudal to the plane indicated for this muscle in the cat, although still near the rostral end of the nucleus. Cells of origin for the adductor muscles were obtained about the obex level being both slightly in front and behind it. The abductor cells of origin were found between the levels in the nucleus for the cricothyroideus muscle and the adductor muscles.

EVIDENCE

These results in the monkey were obtained by the use of two different techniques. The first consisted in observing the chromo-

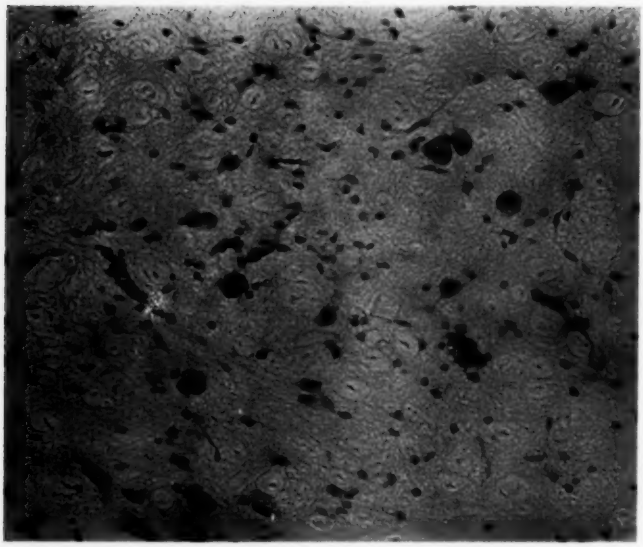

Fig. 2.—Motor neurones in the nucleus ambiguus of the monkey showing chromotolysis. Magnification approximately 90 x.

tolysis in the cells of the nucleus ambiguus after section of the posterior branch of the left recurrent laryngeal nerve. The monkey's larynx was examined by direct laryngoscopy before and after section of the nerve while the animal was under light ether inhalation anesthesia. The animal was allowed to live for two weeks. He was then sacrificed and the brain perfused with formalin and stained with thionin. (Thionin is a differential stain for Nissl granules.) After section of a nerve fiber such Nissl granules disappear producing the condition called chromatolysis. The portion of the nucleus which gave rise to the sectioned fibers of the recurrent laryngeal nerve had cells which were either void of Nissl granules or contained ones poorly stained.

The second technique which must be regarded as more conclusive was directed to the location of the cells of origin in the nucleus ambiguus by direct faradic stimulation. Ether anesthetized monkeys were placed in the McCulloch modification of the stereotaxic instru-

ment after preliminary direct laryngoscopy. A monopolar electrode was then inserted into the nucleus ambiguus according to the tables of D. Altas and W. R. Ingram.[2]

Before placing the monkey in the stereotaxic instrument, the larynx was exposed in the neck in order to obtain a direct view of this organ and especially the cricothyroideus muscle. This was accomplished without interfering with the peripheral nerve supply of the larynx. It was necessary to modify the readings for the monkeys used but ultimately stimulation with favorable readings produced a contraction of the cricothyroid muscle. The trachea was then opened in the midline ventrally to obtain a view of the vocal cords from below. Upon repeating the same stimulation, there was contraction of the cricothyroid muscle but no lateral or medial displacement of the vocal cords. The needle was then moved 1 mm caudoventralward from the first reading (which produced cricothyroid contraction) and the stimulus repeated. It was now evident that adduction of the vocal cord on the side of the stimulus could be obtained but that no movement of the cricothyroid muscle occurred at this level. A lesion was then placed at this last site. The lesion was obviously larger than the area stimulated but it was definitely in the nucleus ambiguus. These results were verified in three monkeys observing both cricothyroid contraction and adduction using approximately the same stereotaxic readings. It is obvious that the difference in position of the needle will vary with the size of the individual monkey.

The level in the nucleus which demonstrated chromatolysis after section of the posterior branch of the recurrent laryngeal nerve was then studied. It was found to be between the sites at which direct stimulation produced cricothyroid muscle contraction and adduction of the vocal cords.

The combined results of these two methods is evidence of a rostralcaudal pattern of innervation in the nucleus ambiguus for the larynx. The cricothyroid is the most rostral of the cells while the adductor muscle group is most caudal.

CLINICAL SIGNIFICANCE

Obviously the presence of such a pattern makes it possible for central lesions to cause impaired function of one or more muscles of the larynx without necessarily an involvement of all of them. Think of the many times we have seen unusual physiologic behavior

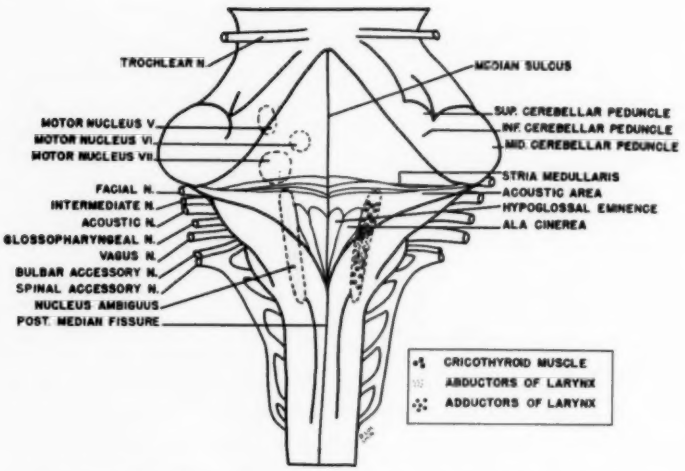

Fig. 3.—Diagram of the pons and medulla of the monkey. Note the shaded area illustrating the nucleus ambiguus. See key demonstrating the approximate location of the motor neurones in the nucleus ambiguus supplying the larynx.

of the larynx with no logical explanation for its occurrence and with despair in our attempts to classify it in accordance with any of our known entities of laryngeal dysfunction. These unusual clinical observations can be definitely attributed to the many cells of origin of motor stimuli which possess specific functions and which for many reasons may be disturbed or handicapped in their action. It is obviously unlikely that a peripheral lesion could be so small and selective as to produce a dysfunction of the isolated laryngeal muscles.

Pressman[3] presented a study of the sphincters of the larynx which established sound reasons for signs and symptoms occasionally observed in central nervous system disease. It created an epoch in our knowledge of the function of the larynx. He reported his observation of a patient with a bilateral recurrent laryngeal nerve paralysis from a central lesion in whom the false vocal cords closed completely with no associated action of the true cords. It is an established fact that this can occur and the anatomical explanation is readily found in the pattern of the motor cells of origin of the superior laryngeal

and recurrent laryngeal nerves in the nucleus ambiguus of the medulla. In the light of our present knowledge of this structure, we may state with confidence that in certain virus diseases, such as bulbar poliomyelitis and degenerative processes of the brain stem, the nucleus ambiguus may be only partially affected unilaterally or bilaterally with a variety of physiologic abnormalities of the false and true cords and the sphincters of the larynx.

CONCLUSIONS

1. A motor innervation pattern for the larynx in the nucleus ambiguus of a monkey has been established.

2. The cells of origin for the innervation of the cricothyroideus, the abductor and adductor muscles have been identified. From a knowledge of their complex pattern a better understanding of the physiological potentialities of the larynx is obtained.

3. Phylogenetically it is logically assumed that a pattern of motor innervation for the larynx in the nucleus ambiguus is present and much more highly developed in man.

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LXXV

LARYNGEAL PAPILLOMATA

A SURGICAL TECHNIQUE FOR THE REMOVAL OF MULTIPLE, RECURRENT OBSTRUCTIVE PAPILLOMATA OF THE LARYNX

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Laryngeal papillomata may occasionally be characterized by being multiple and persistent in their recurrences following their repeated removal. At times they recur to the point of complete obstruction of the glottis necessitating tracheotomy. At other times the repeated removal of the growths perorally has resulted in webbing, scarring, and stenosis of the glottis.

The treatment of this type of papillomata which persists and shows no let-up in momentum may result in glottic scarring and stenosis which requires frequent treatments via bouginages—which is economically hard on the patient and often ends up without permanent cure.

The case being presented is typical of those that have persistent multiple papillomata. This finally resulted in stenosis and obstruction of the glottis to the point of needing tracheotomy.

REPORT OF A CASE

Mrs. A. P., aged 35, had a history of fourteen hospital admissions for the removal of laryngeal papillomata via laryngoscopy since 1942.

The pathological reports of tissues removed were always laryngeal papillomata with no evidence of malignant changes. (Altmann and Basek in a recent survey about to be published, studied 51 cases of laryngeal papillomata from the Columbia-Presbyterian Medical Center and classified the histological characteristics of those cases which turned malignant.) A photomicrograph of a typical specimen obtained from this case is shown in Figure 1.

When first seen by me the patient was dyspneic and the glottis was filled with growths hiding any view of the true or false cords.

Fig. 1.

With the laryngoscope it was difficult to determine where the exact origin of the growths were located. A drawing made from a photograph of an indirect view of the obstructed glottis as seen at that time is shown in Figure 1.

A tracheotomy was done February 17, 1954. Three weeks later the following procedure was carried out: A horizontal collar incision was made about 1 cm above the tracheotomy stoma. The latter had been placed between the first and second rings of the trachea. The midline of the larynx was exposed. A typical fissure incision was made starting at the thyroid notch down through the periosteum continuing as far down as the cricothyroid membrane. The periosteum was elevated and lateralized about one-quarter inch over each ala. The cricothyroid membrane was incised horizontally in its midpart. With a circular electric saw, the midline of the cartilage was cut through (Fig. 3). The alae were then retracted and a large polypoid mass exposed (Fig. 4). This mass measured 1 x 2 cm in diameter. It appeared to be attached mainly to the right side of the larynx. The growth on the right was removed by submucosal resection of the mucosa over the anterior two-thirds of the right true cord, retaining intact submucosal tissues (Fig. 5). With this mass removed, the left side of the larynx was seen to have polypoid tissue

Fig. 2.

involving an even wider area than on the right, though not mounded up as high as that on the right side. The whole involved area on the left side was removed in block, by elevating the periosteum on the inner side of the left thyroid ala, removing both the true and false cords from the anterior commissure back to the vocal process as shown in Figure 6. Bleeding was controlled with cautery.

An acrylic mold (Fig. 7) was then placed with the ring fitted over the tracheotomy tube to hold it in place. The mold used is a modification of molds developed by Goodyear, Robb, Woodward, and Som,[1-4] with modifications by the author. The fit was snug. Molds should vary in length according to the rings involved in the tracheal stoma. It is recommended that they be colored to contrast against body tissues. They should have an airway completely through their axis as suggested by Woodward, be impregnated with radio opaque material as recommended by Robb. Som has more recently reinforced the ring end of his molds with a wire ring. The hole at the grasping end should be in the midline and large enough to be readily grasped with forceps so that it can be rotated when removed perorally.

The alae cartilages were brought together with atraumatic chromic sutures, muscle layers were brought together with interrupted gut sutures. The cricothyroid membrane was sutured with fine

Fig. 3.

Fig. 4.

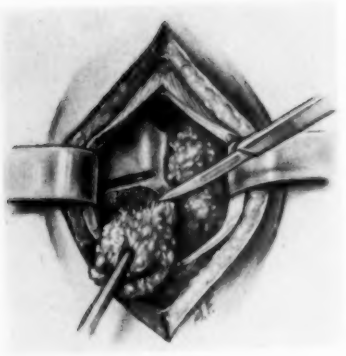

Fig. 5.

Fig. 6.

Fig. 7.

catgut. The skin was closed with interrupted atraumatic dermalon without a drain.

Eight weeks later the acrylic mold was removed perorally. The glottis was found smooth and covered with normal appearing mucosal lining. A drawing made from a photograph taken of an indirect view of the glottis four months after removal of the acrylic mold is shown in Figure 8.

The left cord area had no motion but the right side had adductor function. The voice was whispered in character and was adequate for communication over the telephone. The airway was larger than

Fig. 8.

normal. Sphincteric action of the upper part of the glottis remained active.

COMMENT

This patient has a diathesis for mucosal pathology. In May 1953 she was admitted to the hospital for treatment of intramucosal carcinoma of the cervix. She was at this time also found to have benign papillomata in the bladder.

Since her operation she has had two growths removed from the trachea. One on August 3, 1954 and the other on November 12, 1954. Both were reported as papillomata. At no time since her operation has there been any evidence of recurrence where new epithelium replaced that which was removed from her larynx over a year ago. It is too early to declare or hope for a cure in this case. This procedure is presented as a means of treating those cases which build up such momentum that obstruction and laryngeal stenosis become imminent.

SUMMARY

A surgical technique via laryngofissure with the use of an acrylic mold has been demonstrated for the treatment of those cases of multiple recurrent papillomatosis which develop glottic obstruction.

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SCIENTIFIC PAPERS OF THE AMERICAN OTOLOGICAL SOCIETY

(Continued from June issue)

LXXVI

THE FETAL AND EARLY POSTNATAL DEVELOPMENT OF THE TYMPANIC RING AND RELATED STRUCTURES IN MAN

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As another completed phase of an inclusive study in the field of otological anatomy, the authors are now able to report on the morphogenesis of the tympanic ring and of certain related structures in man. Earlier articles in the series dealt with the following subjects: the vestibular fenestra (oval window) and the fissula ante fenestram;[5,6] the otic capsule, the pericapsular tissues and the pneumatic spaces;[4,8] the cochlear aqueduct and the periotic duct;[9] the tympanic wall of the lateral semicircular canal (that is, the region of surgical fenestration);[2] the cranial aperture of the vestibular aqueduct and the related fovea for the endolymphatic (otic) sac;[3,11,18] the cochlear fenestra (round window) and secondary tympanic membrane;[12] and the auditory ossicles.[1,4,17]

The present article will be devoted chiefly to consideration of the major steps in formation of the tympanic ring. Less detailed information will be presented on the concurrent development of the tympanic membrane, the tympanic cavity, the external acoustic meatus, and the squamous and petrous parts of the temporal bone.

From the Department of Anatomy of the University of Wisconsin and the Department of Anatomy of Northwestern University Medical School (Contribution No. 616 from the latter). A study conducted with the aid of grants provided by the Central Bureau of Research of the American Otological Society, by the Wisconsin Alumni Research Foundation and by the Bess G. Heath Trust Fund Scholarship (University of Wisconsin).

MATERIALS AND METHODS

The tripartate character of the temporal bone at birth is, of course, known to students and practitioners, since this feature is regularly described and figured in familiar texts in anatomy.[13-16] It will become evident to the reader that certain details of morphogenesis have required elaboration.

The current report is based upon a study of microscopic sections in the Otological Collection at the University of Wisconsin.

Six selected stages are illustrated by photomicrographs (Figs. 1, 2 and 3): fetuses of 28 mm (8½ weeks), 36 mm (9 weeks), 55 mm (10½ or 11 weeks), 126 mm (16½ weeks), 190 mm (22 weeks), and 315 mm (34 weeks); an infant five months old. Inclusive microscopic fields were taken at a magnification of 16 diameters; for the purpose of demonstrating detailed histological structure, eleven photomicrographs were taken at higher magnification (57, 100 or 250 diameters).

Reconstructions were prepared from fetal series of 36 mm (9 weeks), 55 mm (11 weeks), 84 mm (12 weeks), 126 mm (16½ weeks), 161 mm (19 weeks). Of these, three are illustrated (Figs. 4 and 5). All of the reconstructions were prepared from Edinger tracings made at a magnification of 20 diameters; the drawings were executed at the natural size of these models. Drawings were prepared, at similar magnification, of the tympanic rings as seen in the reconstructions (Figs. 6a to 6e) and of an actual specimen of temporal bone in a newborn infant (Fig. 6f).

The rings and related structures in the newborn, in two infantile specimens, an adolescent and an adult were drawn at six times natural size (Figs. 7 and 8).

All figures were reduced in reproduction.

OBSERVATIONS AND COMMENTS

28-mm fetus. In the fetus of approximately 8 weeks, the tympanic ring is represented by a poorly defined area of mesenchymal condensation, which first becomes apparent in the region immediately inferior to the developing squama of the temporal bone. This squamous plate is situated lateral to the auditory ossicles (Fig. 1a), and is already definitely formed in membrane bone in the 8-week fetus

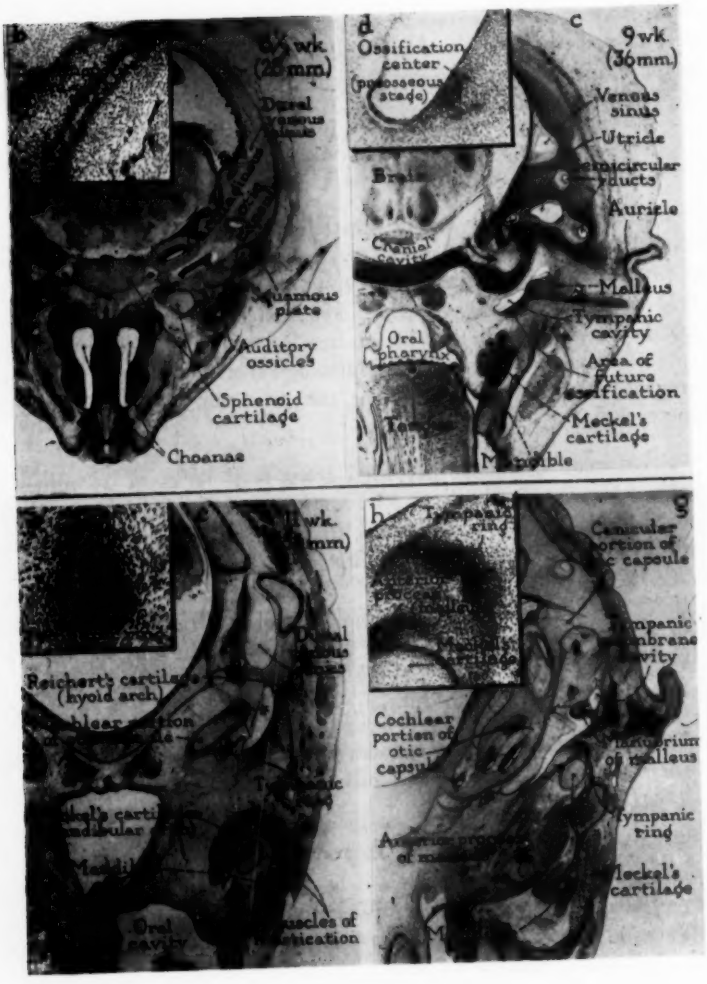
Squamous plate
Auditory ossicles
Sphenoid cartilage
Choanae
Ossification center (perosseous stage)
9 wk. (36 mm.)
Venous sinus
Utricle
Auricle
Malleus
Tympanic cavity
Oral pharynx
Area of future ossification
Meckel's cartilage
Reichert's cartilage (hyoid arch)
Tympanic ring
Cochlear portion of otic capsule
Tympanic ring
Meckel's cartilage

Fig. 1.—Photomicrographs of transverse sections of the head in fetal specimens, depicting histological features in the region of the future tympanic ring. Figs. 1a, 1c, 1e and 1g, X7; Figs. 1b, 1d, 1f and 1h, X44.

a.—In the fetus of approximately 8 weeks, although the squamous plate is readily identifiable as osseous tissue, the tympanic ring is represented merely by a concentration of mesenchyma. b, showing the details of structure of the squamous plate, which is already formed in bone.

c.—In the 9-week fetus a small nodule of preosseous tissue appears in the area of mesenchymal concentration (near the tympanic cavity). This small mass will form part of the body (anteromedial extremity) of the future tympanic ring. The following related structures are also included in the field: oral pharynx; tympanic cavity; manubrium of the malleus; mandible; otic capsule at the transverse level of the tympanomeningeal hiatus (at arrow); brain; cranial cavity with developing meningeal tissue. d.—Detail of histological structure of the body of the tympanic ring (preosseous tissue).

e.—The portions of the body of the osseous tympanic ring, in the 11-week fetus, formed by the secondary centers of ossification, together with the following related elements; the streak of condensed mesenchymal tissue, between the anterior and posterior limbs of the ring, which is the forerunner of the fibrous layer of the tympanic membrane; the tympanic cavity (at asterisk); the plate (at arrow) which, when cleft, will form the innermost part of the external acoustic meatus. f.—Detailed structure of the posterolateral part of the ring, showing the activity of osteoblasts.

g.—Showing the primary ossification center in the head of the tympanic ring, the osseous anterior process of the malleus and Meckel's cartilage. h.—Detailed histological structure of the ossification center, seen at lower magnification in g.

(Fig. 1b). The auditory ossicles and Meckel's cartilage (the first branchial arch) are still composed of young cartilage cells; the incus and stapes are independent of the branchial skeleton, but the malleus is still broadly continuous with Meckel's cartilage (compare 10-week stage, Fig. 4a).

Although the otic capsule, like the auditory ossicles, is in an early stage of cartilaginous development, several structures already osseous are present in the region of Meckel's cartilage; one of these is the bony mandible, situated lateral to Meckel's cartilage; another is the anterior process of the malleus,[1,4,17] which appears as a wholly separate rod of membrane bone at the periphery of the perichondral layer of Meckel's cartilage (see Fig. 1h, 55-mm stage).

36-mm fetus. In the fetus of 9 weeks, an ossification center for the head of the tympanic ring has just appeared; on the contrary, the body of the ring is nothing more definite than an area of concentrated mesenchymal tissue (Figs. 1c and 1d). The forerunner of the ring is insignificant in size in comparison with the otic capsule or with the related mandibular (first branchial) arch.

The cartilaginous otic capsule is traversed by a broad tympanomeningeal hiatus (Fig. 1c, at arrow), between the cochlear and canalicular portions of the capsule. The authors have already described and figured the developmental steps taken in the closure of this channel, and in the consequent formation of the periotic duct, the cochlear fenestra and the fossula by which the latter window communicates with the bay of the tympanic cavity.[12]

50-mm fetus. In the fetus of 10 weeks, following a growth-period of a single week, the tympanic "ring" is a C-shaped combination of four or more ossification centers and intervening zones of condensed mesenchymal tissue which are destined to form additional bone (Figs. 4a and 4b; also Fig. 6a). A large, primary center, which might suitably be termed the head (already present in the 36-mm stage) is located just inferior to Meckel's cartilage where the latter is continuous with the malleus. The small secondary centers are arranged in annular form within the preosseous mesenchymal tissue which will give rise to the body of the ring. Beyond the lowermost of these centers the concentrated tissue extends cranialward in the direction of impending growth.

The partly ossified tympanic ring now lies between the first and second branchial arches (Meckel's and Reichert's cartilages, respec-

tively), posterior to the developing mandible, anterior to the incus, inferior to the squama of the temporal bone. The ring thus lies on the lateral aspect of the expanding tympanic cavity and anteromedial to the ectodermal plate which already shows initial excavation for ultimate production of a relatively spacious external acoustic meatus (compare 11-week stage, Fig. 4c).

The otic capsule, like the remainder of the cranium, is still wholly cartilaginous; so, also, are the auditory ossicles, in which ossification centers will not appear until the 16-week (120-mm) stage has been reached. With matching precociousness, the mandible is already formed in bone.

55-mm fetus. In this slightly older stage (specimen of 10½ or 11 weeks) the secondary ossification centers of the body of the tympanic ring have enlarged and fused, to become continuous with the primary center, or head of the ring (Fig. 4c; also, Fig. 6b). The superior extremity, or head, lies beneath the first branchial arch, near the anterior process of the malleus; the inferior extremity of the body skirts the second branchial arch.

In cross-section the outline of the posterior limb is approximately circular (Figs. 1e and 1f). The anterior limb is lunate (Figs. 1g and 1h). Throughout the extent of the ring, osteoblasts are active (Figs. 1f and 1h).

The tympanic mucous membrane now extends beyond the manubrium of the malleus; the epithelial (ectodermal) plate lies just lateral to the tympanic cavity (Fig. 1e, at arrow).

84-mm fetus. Growth of the tympanic ring has progressed so rapidly that within a brief period of two weeks, between the 50-mm stage (of 10 weeks) and the 84-mm stage (of 12 weeks), the diameter of the ring has doubled (Figs. 6a and 6c).

126-mm fetus. In the fetus of 16½ weeks, bone-formation is active at both the posterior (canalicular) and anterior (cochlear) segments of the ring (Figs. 2b and 2c, respectively); the former segment (Figs. 2a and 2b) remains much smaller than the latter portion (Figs. 2a and 2c; see also Fig. 6d) and retains its roundish cross-sectional outline.

The manubrium of the malleus is lodged in the fibrous stratum of the three-layered tympanic membrane (Fig. 2a); the cuticular

a
Utricle
Vestibule
Saccule
Malleus
Tympanic ring
Auditory tube
b
Tympanic ring (posterolateral segment)
Osteoblasts
c
Osteoclasts
Tympanic ring (anteromedial segment)
Osteoblasts
d
Growing cartilage
Tympanic ring
Facial nerve
Long crus of incus
Manubrium of malleus
Tympanic cavity, membrane
Tympanic ring
Tensor tympani
e
Tympanic ring
Tympanic membrane (trilaminar)
Tympanic cavity
External acoustic meatus

Fig. 2.—Photomicrographs of transverse sections from otological series, continued. Showing further stages in the development of the tympanic ring and of such related features as the anterior process of the malleus, the first branchial arch, the tympanic cavity and the epithelial plate, which is the forerunner of the external acoustic meatus. A to c, fetus of 126 mm (16½ weeks); d and e, fetus of 190 mm (22 weeks). Figs. 2a and 2d, X7; Figs. 2b, 2c, X125; Fig. 2e, X44.

a to c.—Recording the assumption of sulcate form by the anterior segment of the tympanic ring and concomitant formation of a definite external acoustic meatus, where the latter's medial wall contributes the outermost layer of the trilaminar tympanic membrane.

a.—The osseous tympanic ring, circular in transverse section in its posterior (canalicular) portion, semilunar in its anterior (cochlear) portion. The related ectodermal plate is now cleft, newly-formed space being external acoustic meatus.

b and c.—Detailed histology of the anterior and posterior segments of the tympanic ring. In the rapidly expanding ring, osteoblasts form a layer on the outer, convex surface, while osteoclasts cover the inner, concave surface.

d.—Showing both the small posterior segment and the larger anterior part of the tympanic ring in the 22-week fetus, in relation to the tympanic membrane and cavity and to the external acoustic meatus. In addition, the following features are illustrated: centers of ossification in the otic capsule; the squama of the temporal bone in the area af ultimate fusion of the tympanic ring with the canalicular part of the capsule; the malleus and incus, which are still lodged in mesenchymal tissue (prior to investment by the tympanic mucous membrane). e.—Detail of structure of the tympanic ring and of the three layers ultimately contributory to the formation of the tympanic membrane.

layer is derived from the medial part of the now split ectodermal plate; the mucosal stratum is contributed by the epithelial lining of the tympanic cavity (compare 22-week stage, Figs. 2d and 2e). The cleft which has appeared in the original ectodermal plate is the primordial space of the external acoustic meatus.

161-*mm fetus.* The diameter of the tympanic ring has increased by three and one-half times in the interval between the stage of 10 weeks (Figs. 4a and 6a) and the 19-week stage (Figs. 5 and 6e). Additionally, the "ring" now approaches the form of a true annulet. Although the extremity of the body is still rod-like in cross-section, the portion near the head is quite deeply sulcate (Fig. 5, insets). On the internal surface of the anterior segment marked osteoclastic activity is evident, with resultant deepening of the tympanic sulcus. At the same time, bone is being laid down on the external surface.

Bone is now present in the ossicles in the form of a perichondral collar; in the head of the malleus this bone is at last fused with the anterior process of the ossicle. At the malleolar extremity of Meckel's cartilage (where the latter branchial bar was broadly continuous with the ossicle), deorganization is in progress, preparatory to conversion of the altered tissue into that of the anterior ligament of the malleus. Within a 5-week period, the cartilage of the originally broad branchial bar will be reduced to a mere remnant lodged in the ligament.

Ossification is well advanced in the otic capsule.[7,10]

190-*mm fetus.* The fetus of 22 weeks shows the following features of developmental advance: enlarged and still actively growing tympanic ring; more capacious external acoustic meatus; advanced stage of ossification in the otic capsule; expanding tympanic cavity (Figs. 2d and 2e). While the tympanic ring is still undergoing enlargement, it is prevented from fusing, posteriorly, with the facing surface of the canalicular part of the otic capsule by the presence of an area of growing cartilage (Fig. 2d).

The otic capsule and the auditory ossicles have reached adult size. However, external deposition of bone will continue in the case of the capsule, and internal histological modification will be a feature in the further history of the ossicles.[1,4,17]

In the tympanic membrane the intermediate, or fibrous, layer is still a relatively thick stratum; internally, the membrane is com-

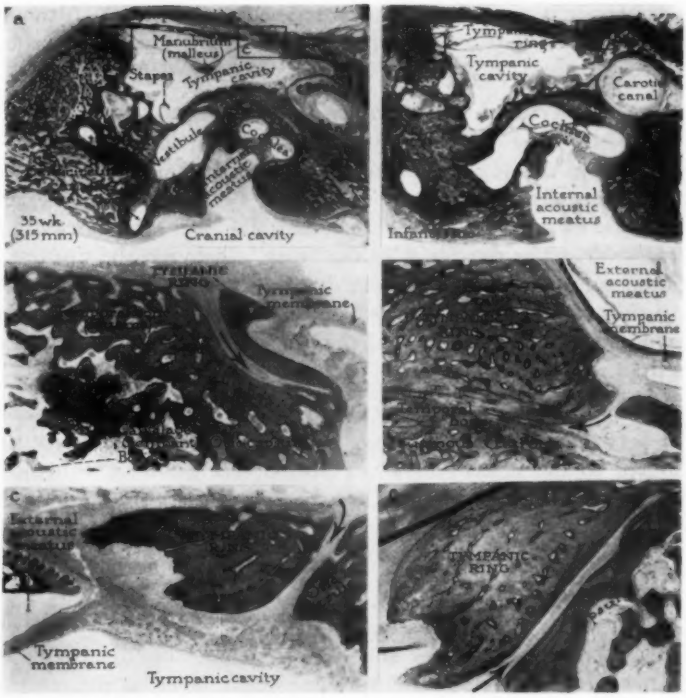

Fig. 3.—Photomicrographs of transverse sections of the head in the advanced fetus and in the young infant, demonstrating fixation of the tympanic ring to the otic capsule. A to c.—Fetus of 315 mm. In a, the area shown at higher magnification in b and c are marked in rectangular blocks; in d, the portions of the entire field picture in e and f are similarly outlined. Arrows indicate "seams" where fusion has not yet taken place (c) or is incomplete (b). Figs. 3a and 3d, X7; Figs. 3b, 3c, 3e and 3f, X25.

a.—In the 35-week fetus, although the posterolateral segment of the tympanic ring is fixed to the squamous plate, a remnant of the cartilage persists. In the anteromedial segment the ring is still free. B and c, demonstrating, at high magnification, the features of histological development at the posterior and anterior aspects, respectively, of the tympanic membrane.

d.—In the 5-month infant, cartilage has been completely replaced by bone; yet a line of cleavage remains, medial to the sulcus for the tympanic membrane, to mark the zone of fusion. E and f.—Recording, in detail, the striking structural difference between the bone of the tympanic ring and of that to which it becomes attached. Arrows (as in b and c) indicate clefts which will subsequently be closed through further growth of bone.

posed of a contribution from the mucous membrane of the middle ear; externally it receives a layer from the epidermal plate. Although the constituent layers are now formed, the membrane itself has not yet become lodged in the *sulcus tympanicus* of the ring (Fig. 2e).

315-mm fetus. In the specimen of 35 weeks, the tympanic ring, although now approaching full dimensions,* remains partly free of attachment to the adjacent portions of the enlarging temporal bone. More striking than the feature of further increase in circumference will be that of lateral extension to become the *pars tympanica* of the *os temporale*. The deficiency in the "ring" (between the tips of the incomplete annulet) will remain in the adult as an indentation termed the *incisura tympanica*. Here, too, the *sulcus tympanicus* will be interrupted, at the attachment of the flaccid segment of the tympanic membrane.

In the 35-week stage, for the first time, localized fusion takes place between the tympanic ring and the squamous plate; fixation is limited to the posterolateral segment of the *annulus*, although a restricted area of cartilaginous growth does persist in the adjacent part of the otic capsule (Figs. 3a and 3b). Fusion at the anteromedial part of the ring has not yet taken place (Figs. 3a and 3c); this step in development is delayed until term.

The three typical layers of bone are well established in the otic capsule. The innermost, or endosteal, stratum, although thin, will remain unaltered; the outermost, or periosteal, layer, as the result of continuing growth, will imbed the otic capsule in so-called petrous part of the temporal bone; the middle layer, still composed of sparsely distributed intrachondrial bone, will be rendered less spongious through the production of endochondral tissue.

Infants. In the newborn infant, the hiatus in the "ring" is closed through fusion superiorly with the *squama* of the temporal bone (Fig. 7a). Additionally, the next step in development is foreshadowed in the presence of a small bony excrescence on the posterior segment of the ring (on the latter's nonarticular aspect)

* Record of growth of the tympanic ring, indicated by increase in diameter: 36-mm fetus, diameter of 1.5 mm; 50-mm fetus, 2.1 mm; 55-mm fetus, 2.6 mm; 62-mm fetus, 2.0 mm; 84-mm fetus, 4.2 mm; 120-mm fetus, 6.15 mm; 126-mm fetus, 6.4 mm; 161-mm fetus, 7.4 mm; 190-mm fetus, 7.9 mm; 210-mm fetus, 8.2 mm; 310-mm fetus, 9.75 mm; 325-mm fetus, 9.5 mm; 350-mm fetus, 10.0 mm; term fetuses (3 specimens), 9.25 mm, 9.25 mm, 10.0 mm; newborn, 10.0 mm; infant of one year, diameter of 10.0 mm.

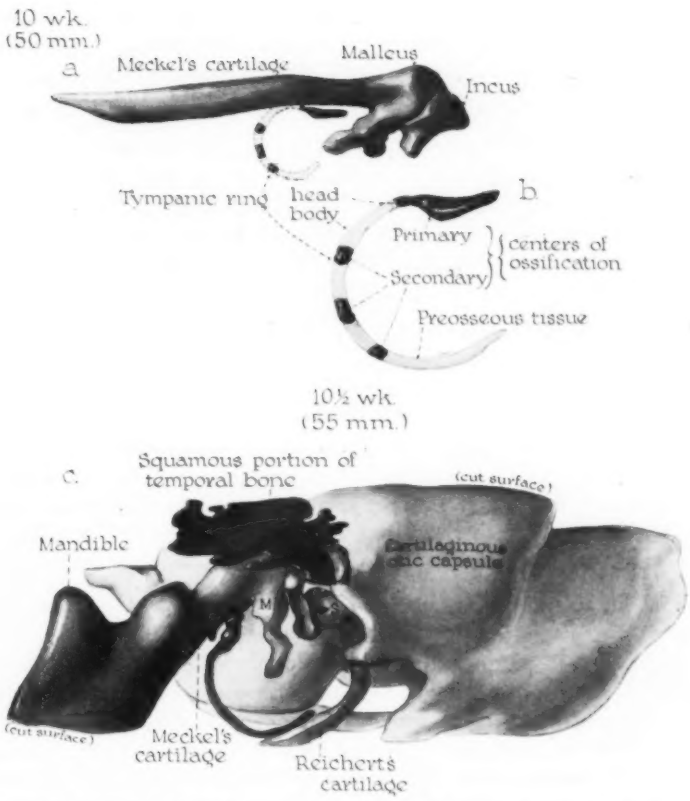

Fig. 4.—Drawings of reconstructions of the tympanic area of the ear, including Meckel's cartilage and the primordial elements of the tympanic ring. Fetal specimenes of 50 mm (10 weeks), and 55 mm (11 weeks). Seen in lateral view. A and c, X 9; b, X 18. Both the osseous areas and the condensed tissue are represented.

a and b.—The auditory ossicles and branchial cartilages of the 10-week fetus in relation to the primary and secondary centers of ossification for the C-shaped tympanic ring.

c.—The tympanic ring, now osseous in structure in the fetus of 55 mm (11 weeks), occupies the interval between the first and second branchial arches (Meckel's and Reichert's cartilages, respectively). Abbreviations: I, incus; M, malleus; S, stapes. The cranial extremity, or head of the "ring" (formed from the primary ossification center) and the anterior process of the malleus are situated inferomedially in relation to Meckel's cartilage, where the latter is continuous with the head of the malleus. The caudal segment of the body (formed from one of the secondary centers) follows the contour of Reichert's cartilage. The ring is C-shaped, being incomplete posteriorly.

and by even more striking evidences of peripheral growth external to the sulcus for the developing tympanic membrane. Fusion has taken place with the petrous portion of the temporal bone (Fig. 7a, at 1, 2 and 3).

On the periphery of the anterior (cochlear) portion of the ring a humplike prominence has appeared (Fig. 6f, at star); upon union of the ring with the facing surface of the squamous plate, this projection will form the superior boundary of the *iter chordae anterius* and will remain as a cranial landmark in the adolescent (Fig. 7d) and the adult.

The areas of fusion of the tympanic ring with the other portions of the temporal bone remain very evident in early postnatal stages. Thus, although attachment has been made in the anteromedial segment, a distinct cleft persists in the zone of anchorage (Figs. 3d and 3f), matching that which still persists in the posteromedial segment (Figs. 3d and 3e). With the attainment of full (that is, adult) dimensions on its internal aspect and fusion circumferentially with the adjacent portions of the temporal bone, enlargement externally is well under way (Fig. 6f).

In the 11-month infant, growth to produce a definitive *pars tympanica* is a striking developmental feature (Fig. 7b). Formation of the bony wall of the external acoustic meatus is continued by the growth of two bony primordia. One of these arises from the posterior segment, the other from the anterior segment, of the tympanic ring. These processes grow toward each other to form the ventral wall of the meatus (Fig. 7b, at small unlabelled arrows).

In a specimen one month older, the projections have just fused at their apices to complete, at that particular point, the ventral bony wall of the external acoustic meatus. The remainder of the future ventral wall is represented by an unossified area (Fig. 7c, at arrow). This hiatus will remain identifiable in the adult, as a shallow fovea on the tympanic wall of the nonarticular part of the mandibular fossa (Fig. 8, near dotted line).

Adolescent and adult. The *pars tympanica* in the skull of the adolescent (13-year-old) is virtually an adult element (Fig. 7d; compare Fig. 7e). The sheath of the styloid process conceals the process itself; deepening of the mandibular fossa means lengthening of the acoustic meatus—to such a degree that the tympanic ring

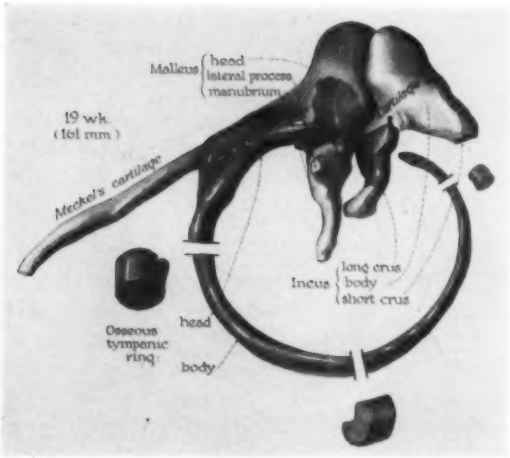

Fig. 5.—Reconstruction of the tympanic ring and related structures, stages concluded; lateral aspect. Fetus of 161 mm (19 weeks). X 8.

The tympanic ring now encloses approximately nine-tenths of a circle. As shown by segments cut at representative levels (and enlarged), the portion of the ring near the head is deeply sulcate, the midportion of the body is somewhat flatter, and the free extremity is virtually circular. The difference in form of the head and body of the ring, established in the 10-week stage (Figs. 4a and 4b), persists here and will persist in postnatal stages.

(now identifiable by the location of the sulcus for the tympanic membrane) is now removed by an appreciable distance from the outer limit of the osseous meatus. The suprameatal spine, prominent in the adult (Fig. 7e), is already definitely formed in the adolescent skull (Fig. 7d).

CONCLUSIONS

In the embryo of 8½ weeks (28 mm) the tympanic ring is represented merely by an area of condensed mesenchymal tissue located just ventral to the first branchial arch (Meckel's cartilage), near the latter's point of continuity with the malleus. Three skeletal structures in this region of the ear are considerably more advanced in development than the ring; they are the mandible, the anterior process of the malleus, and the squama of the temporal bone.

Fig. 6.—Drawings of the tympanic ring, prepared from five reconstructions and one specimen, in successive stages of development, from its first appearance in the fetus of 10 weeks (50 mm) to that of attainment of adult dimensions. X 8 natural size.

In the two-week period between the 10-week and 12-week stages (a and c, respectively) the diameter of the ring is doubled and increased by approximately two and one-half times in the interval between the 12-week stage and birth (c and f, respectively). The "ring," as such, is never a complete annulet; the deficiency is closed through fusion with the *squama temporalis* (see Fig. 7a).

In f the star marks the prominence which persists in some adult skulls as the superior boundary of the *iter chordae anterius* in the squamotympanic (or petrotympanic) fissure. The position of the prominence in the cranium of the adult (see Fig. 8) serves topographically to record the extent of anterior growth, in being separated by a relatively great distance from the outer orifice of the external acoustic meatus. In this figure, portions of the *squama* and the *pars petrosa* are shown in their relation to the *annulus tympanicus*.

In a slightly older specimen (9 weeks, or 36 mm), a small nodule of preosseous tissue makes its appearance in the mesenchyma lateral to the mucous membrane of the tympanic cavity, and in relation to the inferior border of Meckel's cartilage; this is the primary ossification center for the head of the tympanic ring.

One week later (in a fetus of 10 weeks or 50 mm) three or four lesser centers have appeared; these nodular collections of developing bone, together with the intervening osteogenic tissue, outline a "model" of C-shaped form for the body of the ring.

Once ossification has been initiated in both the head and body of the ring, differentiation and growth proceed at a rapid rate—with such celerity, in fact, that within a period of less than a week (in the fetus of 11 weeks, or 55 mm) the ring is a semicircle of bone, the diameter of which is almost double that of the preosseous primordium in the preceding specimen (50-mm fetus). The fundamental relationships are concurrently established: the extremities of the ring skirt the facing surfaces of the first and second branchial arches (Meckel's cartilage and Reichert's cartilage), the superior extremity, or head, being located just anterior to the malleus; the concavity of the C is directed posteriorly and somewhat superiorly; the head lies just lateral to the anterior process of the malleus, which remains independent of the ossicle until the nineteenth week of fetal life (that is, in the 161-mm stage).

In the fetuses of 12 weeks (84 mm) and 16½ weeks (126 mm) the several structures exhibit progressive growth, the diameter of the ring in the latter stage being almost three times as great as the corresponding dimension in the 55-mm fetus. These rapid strides are made in less than five weeks. On the contrary, ossification has not yet been initiated in the otic capsule or in the auditory ossicles of this specimen (but have just made their appearance in the others of approximately the same age). The tympanic cavity has not extended to the farthermost portion of the tympanic ring; the epidermal ingrowth is cleft, the medial wall of the cleft becoming the cuticular layer of the trilaminar tympanic membrane.

Within less than a four-week period, between the fetal stages of 16½ weeks (126 mm) and 19 weeks (161 mm), the diameter of the ring increases by one and one-half times, to enclose nine-tenths of a circle. The sulcus on the inner aspect of the tympanic ring is deep

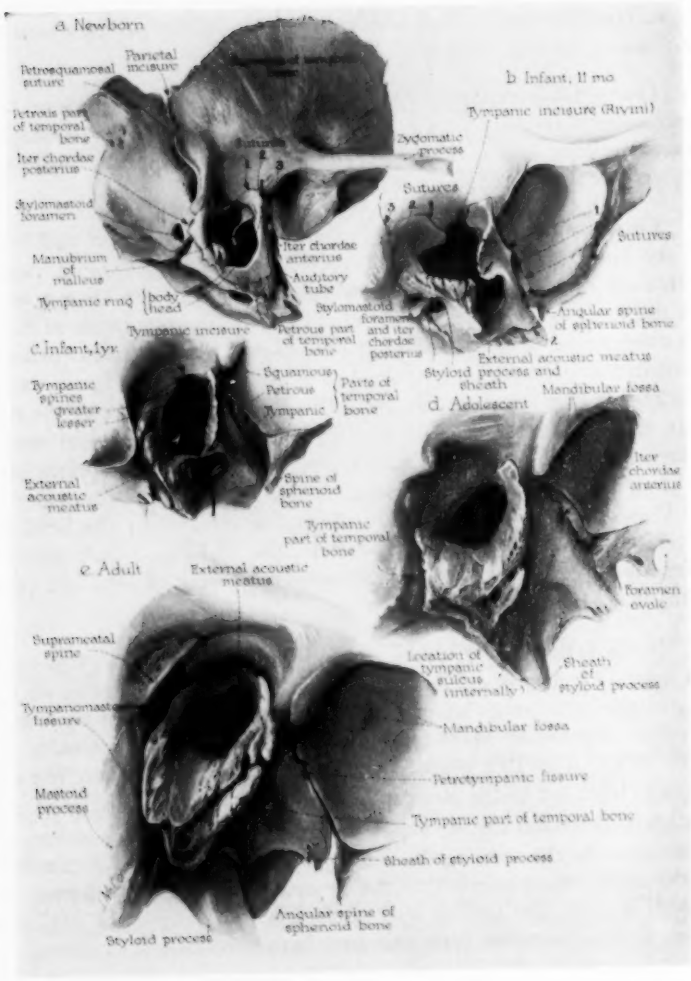
a. Newborn
Petrosquamosal suture
Parietal incisure
Petrous part of temporal bone
Iter chordae posterius
Stylomastoid foramen
Manubrium of malleus
Tympanic ring
body
head
Iter chordae anterius
Auditory tube
Tympanic incisure
Petrous part of temporal bone
b. Infant, 11 mo.
Tympanic incisure (Rivini)
Zygomatic process
Sutures
Sutures
Stylomastoid foramen and iter chordae posterius
Angular spine of sphenoid bone
External acoustic meatus
Styloid process and sheath
Mandibular fossa
c. Infant, 1yr.
Tympanic spines greater lesser
Squamous
Petrous
Tympanic
Parts of temporal bone
External acoustic meatus
Spine of sphenoid bone
d. Adolescent
Iter chordae anterius
Tympanic part of temporal bone
Foramen ovale
e. Adult
External acoustic meatus
Suprameatal spine
Tympanomastoid fissure
Mastoid process
Location of tympanic sulcus (internally)
Sheath of styloid process
Mandibular fossa
Petrotympanic fissure
Tympanic part of temporal bone
Sheath of styloid process
Angular spine of sphenoid bone
Styloid process

Fig. 7.—Drawings of specimens of temporal bone, demonstrating the size and form of the tympanic ring and related structures in the newborn, in older infants, in the adolescent and in the adult, with special reference to the areas of suture established by the tympanic ring and to the mechanism by which the ring is moved to a position at the medial aperture of the internal acoustic meatus. Viewed from either the lateral or inferolateral aspect. X 1.5 natural size.

a.—In the newborn, while the tympanic ring is still an annulet fully exposed on the side of the cranium, it is already anchored, directly or indirectly, to other portions of the temporal bone, at the following lines of suture: squamotympanic (at 1); petrotympanic (at 2); petrosquamous (3). Concurrently, downward growth (with fusion to the *pars petrosa*) forecasts formation of the extensive plate of bone which will be the nonarticular, posterior, part of the mandibular fossa. Both the posterior and anterior openings of the channel for the chorda tympani remain in superficial position, the anterior *iter* being situated just inferior to a small nodule of bone on the outer circumference of the ring which remains identifiable in many adult skulls. On the inner circumference a projection (at unlabelled arrow) represents the beginning of a process which will divide the single aperture of the primordial ring into two portions (see b and c).

b.—In the infant of 11 months, accompanying overall growth, a secondary projection on the inner circumference of the ring presses toward the primary elevation; the latter is now enlarged to assume the form of a crest (each prominence marked by an unlabelled arrow). As a result of growth, the posterior part of the developing *pars tympanica* has begun to cover the styloid process as the latter's sheath, while the anterior part is forming the nonarticular area of the mandibular fossa. The stylomastoid foramen and the posterior channel for the chorda tympani (*iter chordae posterius*) are still separate canals. The sphenoid bone is in contact with the petrous part of the temporal bone (at 4) at the site of the petrotympanic fissure in the skull of the adult (see e).

c.—In the infant one year old, the facing spurs of growing bone (at small, unlabelled arrows) have met to divide an enlarging, and now elongate, channel into two portions; the cranial part (which is the more capacious of the pair) is the osseous external acoustic meatus, while the caudal subdivision (at the large, unlabelled arrow) is a fault in the developing petrous part of the temporal bone which is in the process of closure. Anteriorly are seen the parts of the temporal bone involved in the mechanism of incorporation of the tympanic ring into the adult temporal bone.

d.—During late childhood, despite closure of the fault in the *pars petrosa* and in spite of enlargement of the *pars tympanica* and concurrent deepening of the mandibular fossa, the site of the tympanic ring (internally) remains identifiable on the posterior wall of the nonarticular portion of the fossa. Similar growth, but in a downward direction, has produced the sheath of the styloid process; furthermore, in spite of growth, a small nodule persists as the cranial boundary of the *iter chordae anterius* (a relationship already established in the newborn, a).

e.—The tympanic part and related portions of the temporal bone in the adult (see also Fig. 8).

near the head; depth decreases progressively toward the slender tip of the body, where the ring is merely rod-like in form.

In the 22-week (190-mm) fetus the osseous ring remains independent of the otic capsule. In the canalicular portion of the otic capsule, the cartilage continues to grow in the region of the squama of the temporal bone. Concomitant changes are taking place in the related structures: bone has appeared in the ossicles; Meckel's cartilage is being converted into the anterior suspensory ligament of the malleus; the cleft in the epidermal plate is now complete; the tympanic cavity matches, in longitudinal extent, the adjacent space of the external acoustic meatus.

Fusion of the tympanic ring with the otic capsule does not take place simultaneously throughout the circumference of the annulus. Actually, fusion of the posterolateral segment precedes that of the anteromedial part by approximately four weeks, occurring in the former in the fetus of 315 mm (35 weeks) and in the latter zone at about the stage of term.

In the fetus near term and in the newborn, the tympanic "ring" is an incomplete annulet fully exposed on the side of the cranium; and, now that adult dimensions of the ring itself have been attained, it has become anchored, directly or indirectly, to other portions of the temporal bone at suture areas which are squamotympanic, petrotympanic and petrosquamous. Downward growth, with concurrent fusion to the *pars petrosa*, forecasts formation of the plate of bone which will be the nonarticular, posterior, part of the mandibular fossa. Both the posterior and anterior openings of the channel for transmission of the chorda tympani remain in exposed position, the anterior *iter* being situated just inferior to a small nodule of bone on the outer circumference of the ring which remains identifiable, at the petrosquamous suture, in many adult skulls. On the nonarticular surface of the lateral segment of the ring a bony projection represents the beginning of a process which will result in the formation of the ventral bony wall of the meatus.

In the infant of 11 months, accompanying general growth, a secondary projection on the inner circumference of the anterior segment of the ring presses toward the primary elevation. Owing likewise to growth, the posterior part of the developing *pars tympanica* has begun to cover the styloid process as the latter's sheath, while the

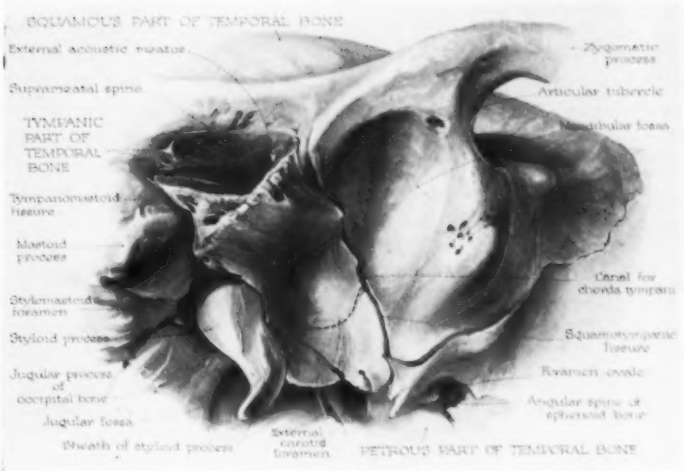

Fig. 8.—The tympanic and related portions of the temporal bone of the adult; specimen pictured in Fig. 7e, viewed from below. X 1.3 natural size.

The part of the *pars tympanica* here seen forms the floor of the external acoustic meatus, the posterior wall of the mandibular fossa and the sheath of the styloid process. The petrotympanic fissure (actually squamotympanic externally) marks the site of original fusion of the tympanic ring with the otic capsule through the *squama* of the temporal bone as an intermediary (see Fig. 7a and compare Fig. 2d). Continuous with the fissure, in the form of a curve concave laterally, a somewhat cribrose streak marks, on the outer surface of the tympanic part of the temporal bone, the site (internally) of the tympanic ring. The distance (1.5 cm, approximately) between this streak (along dotted line) and the lateral free margin of the *pars tympanica* represents the extent of the latter's growth in late fetal and postnatal stages.

anterior part is forming the nonarticular portion of the mandibular fossa.

In the infant of one year the facing spurs of growing bone have met to encircle at that point the bony ventral wall of the external auditory meatus. The remainder of the ventral wall of the meatus is still preformed in membrane. The lateral part of this membranous area represents the ventral wall of the greater part of the ultimate external auditory meatus. The other area is slow to ossify, but when ossified, completes the medial portion of the wall of the external

auditory meatus; at the same time its external surface forms the nonarticular portion of the mandibular fossa. In some adult skulls the small area last to ossify is identifiable as a shallow fovea, the medial edge of which marks the site (internally) of the anterior attachment of the tympanic membrane. Thus the two bony spurs, first fused at about the age of one year, spread to produce the complete anteroventral bony wall of the meatus, the nonarticular part of the mandibular socket, the sheath of the styloid process and the plate of bone which underlies in part the apical portion of the petrous bone and forms part of the wall of the carotid canal.

The original tympanic ring, as seen at term, will remain, in the adult, at the deep end of the external auditory meatus as that portion of the bony meatal wall which supports the tympanic membrane.

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The reconstructions were prepared at the University of Wisconsin by Paul E. Poenisch, with the technical assistance of Mildred Fuller. At Northwestern University Medical School the photomicrographs were taken by Madge Walsh, labelled by Rosamond Howland; the drawings of the reconstructions and of the specimens were executed by Jean McConnell.

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LXXVII

CONGENITAL ATRESIA OF THE EAR IN MAN AND ANIMALS

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In a previous report[1] the anatomical findings were reviewed in all the cases of so-called congenital atresia of the ear where the temporal bones were sufficiently well described to permit a detailed developmental analysis of the findings. Altogether 59 temporal bones, belonging to 50 individuals, could be utilized. According to the severity of the malformation, the cases were divided into three groups.

In the first group, the malformations of mild degree, either the external auditory meatus, although hypoplastic, or at least its inner part is present. The tympanic bone is hypoplastic and somewhat misshapen and the drum membrane smaller than normal. The tympanic cavity is normal in size or hypoplastic. Its contents show malformations of varying degree. This group comprised 17 temporal bones.

The majority of the known cases belongs to the second group, the malformations of medium degree. The external auditory meatus is completely absent, the tympanic cavity is, as a rule, diminished in size and its contents deformed in varying degree. The lateral wall of the tympanic cavity forms the so-called atresia plate which is either completely or partly osseous. The tympanic bone is present in some cases and missing in others. If present, it is badly misshapen, often platelike; frequently it contains at its center a membrane-like mass of connective tissue which is continuous with the connective tissue occupying the place of the missing external auditory meatus.

Regardless of whether a malformed tympanic bone is present or not, the upper end of Reichert's cartilage might show anomalies.

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It might be abnormally situated and hyperplastic and might form the main part of the atresia plate. If the tympanic bone is defective, outgrowths are formed from the squama and from the floor of the tympanic cavity which extend downward and upward respectively and diminish or close the defect of the lateral wall. This group contained 32 temporal bones.

In the third group, the malformations of severe degree, the external auditory meatus is again absent and the tympanic cavity either markedly hypoplastic or completely missing. This group contained ten temporal bones.

In spite of the greatly increased interest in the operative treatment of congenital atresia very little has been added during recent years to our knowledge of the anatomical changes in these conditions. Only one case with a bilateral atresia was examined histologically.[22] Both temporal bones belong to the medium degree of the malformations; Reichert's cartilage was not hyperplastic and the atresia plate was formed by processes from the squama and from the tympanic floor.

Another case was observed lately in Presbyterian Hospital.

REPORT OF A CASE

D. C.: The parents of the patient were 26 and 28 years old, and in good health. There was no history of malformations in the family. The first pregnancy of the mother was uneventful; labor occurred one week before the expected date and delivery was uncomplicated. The birthweight was 2160 g, the length 44 cm.

The child showed multiple malformations. Most notable was a lateral and downward slanting of the axes of both palpebral fissures ("antimongoloid obliquity") with symmetrical small colobomas in the outer parts of the lower lids; this was associated with a marked hypoplasia of the mandible with the tip of the latter 2 cm behind that of the maxilla. The mouth could only be opened 1.5 cm in a vertical direction. The tongue was set back with the mandible; it appeared to impinge on the nasopharynx and almost to obstruct it. The configuration of the cheeks was normal.

The bridge of the nose was broad and flattened, the eyes were wider apart than usual (hypertelorism). The distance between the right and left inner canthus was 2.5 cm.

The auricles showed normal location and size, but slight anomalies of their shape; the openings of the external meatuses were well developed, but the canals seemed to end blindly about 5 mm from the entrance.

There were multiple capillary hemangiomas in the skin of the face and the scalp.

Clinical and x-ray examination revealed numerous other skeletal deformities: Bilateral absence of the condyloid and coronoid processes of the mandible and of the glenoid fossa of the temporal bone; hypoplasia especially of the radius and to a lesser extent of the ulna, more marked on the left than on the right side; absence of the first metacarpal bone and of the thumb on both sides; abnormal shortness of both first toes; hypoplasia of both ilia and increased slope of the acetabular roofs.

Clinical course. The child tended to accumulate mucus in her throat and had difficulty in swallowing it. She was unable to take fluids by mouth normally and had to be fed by nasogastric tube. She lost weight gradually and in her third month of life had increasing respiratory difficulties and attacks of coughing. A few weeks later she died of lobular pneumonia.

No other malformations than the ones mentioned above were seen at autopsy.

HISTOLOGICAL EXAMINATION OF THE TEMPORAL BONES (VERTICAL SERIAL SECTIONS)

Right side:

External ear. The auricle is not contained in the specimen. The external auditory meatus ends blindly. The place where its deeper part is usualy located is filled with connective and fat tissue. The parotid gland is not found in its usual place.

Middle ear and eusatchian tube. Only the osseus portion of the tube is seen in the sections. It shows a relatively wide lumen and normal structure.

The tympanic cavity, i.e., the space contained within the bony or membranous walls, has a greatest length of about 10 mm, height of 11 mm and width of 4 mm. The antrum is relatively narrow and measures 4.5 mm in height and 3 mm in width. Compared

with the tympanic cavity of the normal newborn, the malformed cavity is almost as long and in the anterior two-thirds of approximately the same height and width. The posterior third and particularly the antrum are considerably narrower. The lumen is well developed and the mucosa thin in most places and covered with cuboidal epithelium. The mucosa shows greater height only in the epitympanic space and in some niches of the walls; it is then lined with pseudostratified columnar epithelium. The window niches are already free from myxomatous tissue and the sinus tympani is well developed. Myxomatous tissue still fills the mastoid antrum but a recessus of the middle ear lumen extends into it. There is still no evidence of pneumatization of the mastoid but pneumatization of the squama has already started.

The middle ear lumen contains homogeneous pink staining masses with numerous round cells, polymorphonuclear leucocytes and some swollen desquamated epithelial cells. The tunica propria of the mucosa shows moderately marked lymphocytic and plasmocellular infiltration of the subepithelial layers which is more marked in areas with higher mucosa, particularly in the epitympanum. Here a partial organization of the exudate with ingrowth of vascular buds, formation of tissue bridges and formation of smaller and larger cysts has taken place.

Osseous walls. In the lateral wall the place of the tympanic membrane is taken by a thin ovoid shaped bony plate which measures 4 mm in greatest length, 7 mm in height and 1.5 mm in average thickness (Fig. 1). This plate is in the greater part of its circumference fused with the adjacent parts of the temporal bone, in the upper and posterior quadrant with the squama and in the lower quadrant with the part of the petrous bone which forms the floor of the middle ear. In a part of the postero-inferior portion of the circumference it is adjacent to an irregularly pyramid-shaped bone which lies between the parts of the squama extending downward behind the bony plate which replaces the drum and the parts of the petrosum which form the petrous portion of the mastoid and the posterior part of the floor of the middle ear (Fig. 2). The bone in question is already extensively fused with the adjacent bones. In front of the anterior quadrant of the bony plate, the lateral wall of the middle ear is membranous for a short distance and still farther forward it is again almost completely formed by bone, by a bony lamella of the tegmen reaching downward toward a bony lamella extending upward from

Fig. 1.—Right side. Section through the windows. The abbreviation ch. indicates chorda tympani; o., ossicular rudiment; r., round window membrane; st.c., anterior crus of stapes; st.f., stapedial footplate; t.h., tympanohyale; te., tegmen and ty., tympanic plate.

the floor. The squama shows otherwise normal configuration. The posterior portions of the lateral wall are formed by processes of the squama and of the petrous bone extending downward and upward respectively which are separated from each other by a narrow strip of connective tissue, the external petrosquamous suture.

The tegmen tympani is completely ossified with the exception of a few narrow vascular channels which connect the middle ear mucosa with the dura of the middle cranial fossa. It shows more or less normal configuration. Laterally, it is separated from the squama by the internal petrosquamous suture. The floor is formed as usual by a bony lamella extending laterally from the lower portion of the labyrinthine capsule.

Fig. 2.—Right side. Section through the posterior part of the tympanic cavity. The abbreviation ch. indicates chorda tympani; o., ossicular rudiment; styl., styloid process; t.h., tympanohyale; t.st., insertion of the tendon of the stapedial muscle and ty., tympanic plate.

On the medial wall the window niches, the promontory, the fissula ante- and the fossula post-fenestram are well developed.

Auditory ossicles, styloid process and intratympanic muscles. The malleus and incus are represented by a single ossicle which lies in the epitympanic space (Figs. 1 and 2). It consists of a body, 2.5 mm high and 1.5 mm thick, and a posterior process which extends for several mm backward towards the antrum. The total length of the ossicle is 4.5 mm. The body is connected through a thin bar of bone with the medial wall of the middle ear; it has, however, no connection with the stapes. The tenosr tympani muscle is well developed. It lies more laterad than usual in a bony canal of the undersurface of the tegmen, approximately at the border between the middle and

lateral third of the latter. Its tendon inserts at the anterior pole of the ossicle. The stapes (Fig. 1) has a normal looking footplate, measuring 1.5 : 2.0 mm. It lies in the oval window and is connected with the window frame through the annular ligament. The crura are severely misshapen. The anterior crus originates from the anterior portion of the footplate; it is a slender, solid rod of bone with a greatest height of 1.75 mm; the capitulum is absent. The posterior crus, instead of inserting on the posterior portion of the footplate, is connected through a kind of syndesmosis with the bone of the postero-inferior circumference of the window niche adjacent to the footplate. It is also a slender rod of bone which runs upward and forward and fuses with the upper third of the anterior crus. The tendon of the stapedial muscle inserts on the lowermost portion of the posterior crus, near its syndesmotic connection with the bone of the window niche (Fig. 2). Since the descending portion of the facial nerve shows an abnormal course, traverses the posterior part of the middle ear and leaves it at a point which is farther forward than the normal stylomastoid foramen, the relationship between the stapedial muscle and the nerve has been changed. The tendon enters the stapedial canal at a point below the facial canal; a pyramidal eminence has not been formed. The stapedial canal is completely filled with the well developed stapedial muscle. The canal lies first below (Fig. 3) and then behind the facial canal but always adjacent to it. Near the point where the facial nerve emerges from the temporal bone, there is a fairly large communication-opening between the facial and the stapedial canal, through which a branch of the facial nerve runs to the muscle. From there the stapedial muscle still extends farther downward, backward and outward. It is very well developed and its diameter exceeds by one third the diameter of the normal stapedial muscle of the newborn. It eventually becomes completely enclosed into a bony canal within the inferolateral portion of the petrous bone which is situated between the enchondral and periosteal layers of bone. It eventually reaches the outer periosteum not far from the petro-occipital synchondrosis. The styloid process is still completely cartilaginous. Its upper end is markedly hypoplastic and ends much farther anteriorly than under normal conditions, just behind the level of the posterior circumference of the oval window. The styloid does not penetrate into the floor of the middle ear but just produces a slight indentation in the overlying irregularly pyramid-shaped bone.

Fig. 3.—Right side. Section through the lower part of the facial canal. The abbreviation c.t.i. indicates infero-medial portion, c.t.s., supero-lateral portion of tympanic cavity; ch., chorda tympani; m.st., stapedial muscle; N.VII., facial nerve and o., ossicular rudiment.

The facial canal is completely closed towards the middle ear. Just behind the oval window the nerve, instead of going backward and downward, turns backward, downward and laterad at the crest of a ridge which protrudes from the posterior wall of the middle ear into the cavity and which divides the lumen into a superolateral portion which communicates with the antrum and an inferomedial portion which ends blindly (Fig. 3). The facial nerve thus leaves the temporal bone more anteriorly than under normal circumstances. Its bony canal is also in this portion completely closed. The pyramid-shaped piece of bone which is intercalated between the postauditory process of the squama and the part of the petrous bone which forms the posterior portion of the floor of the middle ear participates in the

formation of the lowermost part of the facial canal. The nerve runs between this bone and the petrous portion laterad and eventually leaves the temporal bone. Near the place where it emerges it gives off the chorda tympani. The latter runs over the lateral surface of the bone in question upward, forward and inward, and is partially surrounded by the superficial layers of the bone (Fig. 3). It enters the tympanic cavity between this bone and the posterior circumference of the bony plate which replaces the tympanic membrane and which is here fused with the postauditory process of the squama. In the tympanic cavity, the chorda runs forward along the inner surface of the plate (Figs. 1 and 2) and leaves the tympanic cavity again just in front of the plate.

The tympanic nerve originates from the glossopharyngeal nerve and runs upward and forward over the promontory. It gives off a few thin branches and is connected with the internal carotid plexus through two branches. The lesser superficial petrosal nerve is absent. The geniculate ganglion and the greater superficial petrosal nerve are normally developed.

The blood supply of the middle ear does not show any anomalies.

The temporomandibular joint is not contained in the sections.

Inner ear. The labyrinthine capsule and the membranous labyrinth do not show any abnormalities.

Left side:

External ear. The findings are the same as on the right side.

Middle ear and eustachian tube. The cartilaginous, as well as the osseous, portion of the tube is contained in the sections; both look normal.

The middle ear lumen has the same width and contents as on the other side, the mucosa an identical structure.

Osseous walls. The place of the drum is again taken by a bony plate which shows the same relationship to the adjacent bones as on the right side. It is, however, somewhat larger. Otherwise the walls show the same relationship as on the other side.

Ossicles, intratympanic muscles and styloid process. The malleus (Fig. 4) is ovoid shaped and measures 3 mm in length, 2 mm in

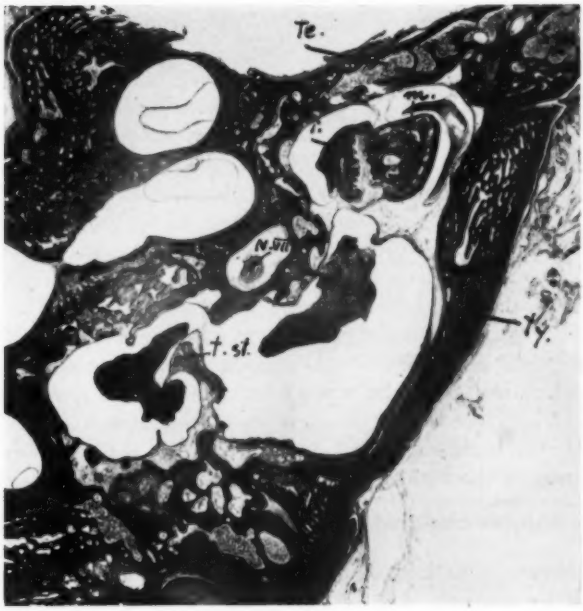

Fig. 4.—Left side. Section through the incudo-malleolar joint. The abbreviation i. indicates incus; m., malleus; N.VII., facial nerve; squ., squama; t.st., tendon of stapedial muscle; te., tegmen tympani and ty., tympanic plate.

height and width. It lies in the epitympanic space and occupies the same position as normally the head of the ossicle. The tensor tympani muscle is well developed and shows the same location as on the other side. Its tendon inserts again at the anterior pole of the malleus. The incus (Fig. 4) is represented by a separate small bone, 3.5 mm long, 2 mm high and 1.25 mm thick; it is connected by a joint with the postero-inferomedial portion of the malleus. Posteriorly it extends, as usual, towards the antrum. It is without connection with the stapes. The stapedial footplate measures 2 mm in greatest length and 1.5 mm in width. In the anterosuperior half of its circumference the layers of cartilage which cover the rim of the oval window and the rim of the footplate are somewhat thicker than usual and, accordingly, the fibers of the annular ligament shorter. In the postero-

inferior half the cartilage of the window is fused with that of the footplate and the annular ligament is absent. The footplate itself is less completely ossified and the layer of cartilage on the vestibular surface somewhat thicker. In the postero-inferior quadrant the margin of the footplate rises to a low crest which is also connected by a continuous layer of cartilage with the adjacent part of the window-niche (Fig. 5). The anterior crus rises from the central parts of the footplate to a height of about 1.75 mm, the posterior crus from the just mentioned crest and unites 0.3 mm above its point of origin with the anterior crus. The common upper end of both crura is slightly thickened but the capitulum itself is absent. The stapedial muscle does not insert on the stapes but on the postero-inferior circumference of the oval window niche, near the footplate. It shows otherwise the same location and topographical relationship to the facial nerve as on the other side. The styloid process and the facial nerve show the same location as on the other side. The chorda tympani, however, is absent. The tympanic nerve shows the same course as on the right side; the lesser superficial petrosal nerve is again missing. The blood supply of the middle ear shows no apparent abnormalities.

The temporomandibular joint is not contained in the sections.

Inner ear. The labyrinthine capsule as well as the membranous labyrinth are normal.

SUMMARY OF ANATOMICAL FINDINGS

Right side. There are slight anomalies of the shape of the auricle, absence of the deeper parts of the external auditory meatus, the tympanic bone plate-like, participating in the formation of the lateral wall of the tympanic cavity ("atresia plate"); there is normal development of the middle ear lumen and hypoplasia of the posterior part of tympanic space and of antrum; the malleus and the incus are represented by a single ossicle connected by a bony bar with the medial wall of the middle ear but not with the stapes; there is lateral displacement and abnormal insertion of the tensor tympani muscle; the crura of stapes are malformed, the posterior crus connects with the wall of the window niche; there is hyperplasia, abnormal location and insertion of the stapedial muscle. The upper end of the styloid process is hypoplastic, not entering the middle ear but producing an indentation in the overlying irregularly pyramid-shaped bone. The descending part of the facial nerve takes an anomalous course

Fig. 5.—Left side. Section through the stapedial footplate and the anterior crus of stapes. The abbreviation an. indicates annular ligament and f. the area of fusion between the footplate and the window niche.

leaving the middle ear more anteriorly than normally. The lesser superficial petrosal nerve is absent.

Left side. The findings are essentially identical, with the following exceptions. The malleus and incus are separate small bones, the latter again without connection with the stapes. The annular ligament is absent in one-half of the circumference of the stapedial footplate; there is an abnormal configuration of the footplate and of the cura and absence of the chorda tympani.

Comment. The malformations just enumerated can be explained as the result of developmental disturbances affecting the posterior ends of the first and second branchial arches and the first branchial cleft and pouch.

Anomalies of the first branchial arch resulted in the malformation of malleus and incus and of the tensor tympani muscle. Anomalies in the formation of the membranous bones adjacent to the upper end of Meckel's cartilage caused the absence of the Folian process of the malleus and the platelike deformity of the tympanic bone.

Anomalies of the upper end of the second branchial arch accounted for the malformation of the stapes and of the stapedial muscle, for the hypoplasia of the upper end of the styloid process, for the presence of the irregularly pyramid-shaped bone which evidently represents a malformed tympanohyale, the ossified uppermost portion of Reichert's cartilage which normally gives rise to the eminentia styloidea and part of the lower portion of the facial canal, furthermore for the abnormal course of the facial nerve which shows a close topographical relationship to the cartilaginous laterohyale and the osseous tympanohyale. The fact that the chorda tympani was present at least on the right side and located within the tympanic cavity, mediad to the atresia plate, indicates that the plate occupies the approximate site of the tympanic membrane. The absence of the chorda tympani on the left side is a relatively rare finding. The fibres usually contained in this nerve are most probably using other routes to get to their destination; the same might be true of the fibres which usually compose the lesser superficial petrosal nerve. The absence of the deeper parts of the external auditory meatus is due to hypoplasia of the primary meatus and aplasia of the meatal plate. The first branchial pouch showed almost normal development.

Of great practical interest is the bilateral stapedial ankylosis, on the right side due to an abnormal connection of the posterior crus to the window niche, on the left mainly due to a partial lack of formation of the annular ligament. In both instances operative attempts at improving the hearing would have been unsuccessful unless a fenestration of the lateral semicircular canal had been performed.

The case in question belongs to malformations of medium degree with the atresia plate formed by the malformed tympanic bone, according to the classification given by me previously.[1]

Associated with the ear malformations were antimongoloid obliquity of the palpebral fissures with colobomas of the lower lids, hypoplasia of the mandible with malformation of both temporomandibular joints, hypertelorism, but not definitely hypoplastic or defective zygomatic arches.

All the described anomalies, with the exception of the hypertelorism, are part of a syndrome first recognized as a distinct clinical entity by Franceschetti and Zwahlen[7] and called mandibulo-facial dysostosis, by others, not quite appropriately, Treacher-Collins syn-

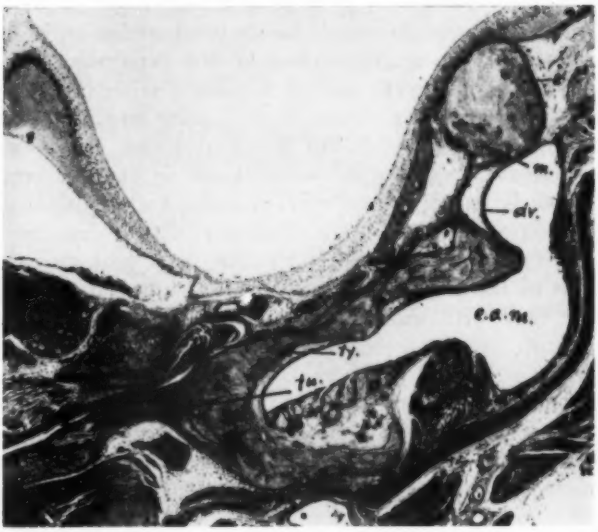

Fig. 6.—Rat D 9124. Right side. The abbreviation dr. indicates drum membrane; e.a.m., external auditory meatus; i., incus; m., malleus; tu., Eustachian tube and ty., tympanic bone.

drome. The just described case does not fulfill all the requirements of the complete or standard type of the condition; it must therefore be grouped with the incomplete forms in which the appearance of the auricle is almost normal but the hearing often impaired. The condition is due to an inhibitory process affecting the facial bones and other derivatives of the first branchial arch. The hypertelorism is evidently a coincidental malformation. It can be one of the symptoms of the craniofacial dysostosis of Crouzon in which atresia of the external auditory meatus is also frequently found (Nager and de Reynier, Wiegand[25]).

Of particular interest is the association of the malformations of the head with those of the extremities (hypoplasia of both radii, to a lesser extent of the ulnae, absence of the thumbs, hypoplasia of both first toes). Such a combination was also observed in one case by Nager and de Reynier who point out that malformations of the extremities, particularly syndactyly, might occur in certain other

malformations of the cranial skeleton (acrocephalosyndactyly-Apert[4]) and in Crouzon's dysostosis (cephalosyndactyly-Vogt[23]). They suggest the term acrofacial dysostosis for the combination of mandibulofacial dysostosis with malformations of the extremities. It seems noteworthy that in Nager's and de Reynier's, as well as in the just described case, defects of certain skeletal parts and not syndactyly were present. Franceschetti and Klein[6] feel that the creation of the subgroup of acro-facial dysostosis is not appropriate because in Nager's and de Reynier's case there was also a cleft palate present. Since the literature shows a high frequency of malformations of the extremities in cases of cleft lip and palate, they think that the malformations of the extremities should rather be linked with those of the palate than with the malformations of the facial skeleton. It must be mentioned, however, that in the just described case neither a cleft lip nor palate was present and that the relative frequency of a combination of atresia of the ear with defects of the radius and the distal parts connected with it in cases with and without cleft palate but without other malformations of the facial skeleton had already been pointed out by Essen-Möller.[5] The combination of atresia of the ear with other malformations in the head region can be regarded as disturbance of induction with subsequent manifestation in various parts of the head. Combination with malformations in other parts of the body could be either accidental or if they occur frequently, such as the just mentioned skeletal defects, the possibility of developmental interrelationships or of pleiotropic gene action should be considered.

The case just described and the one reported by Rüedi bring the total number of usable observations to 63 temporal bones from 52 individuals; 36 of them belong to the malformations of medium degree.

It has been known for a long time that similar malformations also occur in animals, such as mice, rabbits, hares, pigs, horses, cattle and sheep.[3] Unfortunately, in most of these cases, there was no anatomical and practically no histological examination of the malformed areas, and very often no mention is made as to whether the auricle alone or the external auditory meatus and the middle ear were also affected.

Of particular interest is Mohr's[20] observation in Norwegian sheep of a combination of agenesia of the ear with cleft palate, shortening

and ankylosis of the mandible and other malformations. This combination of developmental anomalies is similar to the dysostosis mandibulofacialis in man.

Histological examinations of cases of congenital atresia in animals seemed highly desirable since they could greatly contribute to a better understanding of the formal genesis of this group of malformations. Under these circumstances, I regarded it as my particularly good fortune that through the courtesy of Dr. J. Gillman (Johannesburg, South Africa), I had on opportunity to examine the heads of five rats with unilateral congenital atresia of the ear. The animals came from mothers which had been injected subcutaneously before and during pregnancy with 1 cc of a watery solution of trypian blue. Before discussing the question of a possible causal connection between the treatment and the appearance of the ear malformations, the findings in the five rats shall be reported:

Rat D 9124, 20 days old. Mother received five injections before and one on the 14th day of pregnancy.

The right auricle is reduced to a small hillock, the external auditory meatus recognizable. The left auricle and external auditory meatus are normal. No other malformations are noted on the head. The brain shows internal hydrocephalus, but otherwise there is normal configuration.

HISTOLOGICAL EXAMINATION OF THE TEMPORAL BONES (VERTICAL SERIAL SECTIONS)

Right side:

External ear. The auricular rudiment shows normal histological structure. The external auditory meatus is narrower than normal. The tympanic bone is hypoplastic and misshapen (Fig. 6). The part which normally forms the tympanic bulla is absent. The drum membrane is much smaller than normal.

Middle ear. The eustachian tube is hypoplastic and ends blindly at approximately the place where it should go over into the epithelial anlage of the middle ear; the latter is completely absent. The tympanic space is small and narrow, slitlike, and is filled with loose connective tissue. It extends backward over the anterior third of the lateral surface of the labyrinthine capsule. Both labyrinthine windows are missing.

The malleus and incus are relatively well developed, particularly the malleus (Fig. 6). They occupy a more ventral position than normally and almost completely fill out the tympanic space. The head of the malleus and the body of the incus abut against the thin tegmen, the handle of the malleus against the inner portion of the tympanic ring, without, however, being fused with these structures. The stapes is completely absent. The tensor tympani and stapedius muscles as well as the styloid process are missing. The geniculate ganglion is well developed. The facial nerve goes in a short canal over the anterolateral aspect of the labyrinthine capsule into the tympanic space. It runs for a short distance along its medial wall backward and downward and then turns laterad and leaves the middle ear space. The chorda tympani is missing. The stapedial artery is well developed and enters the middle ear space just behind the place where the facial nerve emerges. It first runs along the medial wall forward and upward and eventually penetrates the base of the skull in a short canal just in front of the labyrinthine capsule. The temporomandibular joint and the mandible are well developed.

Inner ear. In the osseous capsule absence of both windows and of the cochlear aqueduct is noted.

The membranous semicircular canals including the cristae are normal. The utricle and the saccule are both markedly dilated and communicate with each other through a large opening. Both maculae are present; the perilymphatic cystern is almost completely obliterated. The endolymphatic duct and sac are missing. No separate cochlear duct is seen; its place is taken by an extension of the dilated sacculus. The thin VIII nerve supplies the cristae and maculae.

The left side shows no abnormalities.

Comment. The malformations of the external and middle ear are again explained by disturbances affecting the posterior ends of the first branchial arch (hfypoplasia of the part of the auricle derived from the first arch, slight anomalies of malleus and incus, absence of the tensor tympani muscle, hypoplasia of the tympanic bone); the second branchial arch (hypoplasia of the part of the auricle derived from the second arch, absence of stapes, stapedial muscle and styloid, abnormal course of the stapedial artery, probably due to the same causes as the absence of the stapes; abnormal course of the facial nerve through the middle ear space, possibly in conjunction with the anomalies of the upper end of Reichert's cartilage); the first bran-

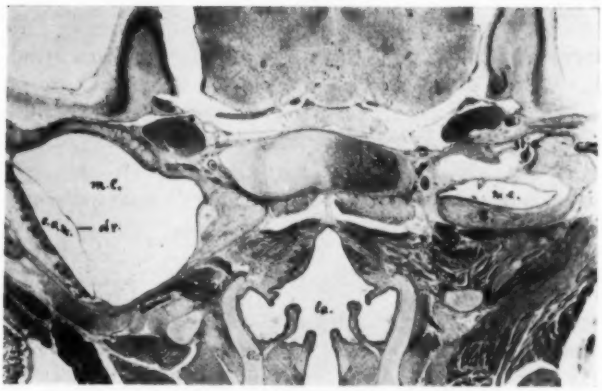

Fig. 7.—Rat E 6376. Section through both middle ears. The abbreviation dr. indicates drum membrane; e., rostral entotympanicum(?); e.a.m., external auditory meatus; la., larynx; m.e., epithelial anlage of middle ear; and st.a., stapedial artery.

chial cleft (hypoplasia of external auditory meatus and drum); the first pouch (hypoplasia of the tube, absence of the epithelial anlage of the middle ear, hypoplasia of the middle ear space in general). The absence of the chorda tympani is, as explained in the preceding case, rare in men, but without any particular significance.

The severe malformation of the membranous labyrinth is the result of a faulty differentiation of the otocyst, probably the consequence of a primary displacement (see comments on rat E 3). The absence of the labyrinthine windows is explained by the absence of the stapes and of the cochlea respectively. It might be mentioned that in man a combination of malformations of the external and middle ear with various anomalies of the inner ear is quite common and occurs in one third of the cases.[2]

Considering the condition of the external auditory meatus, the case just described would belong to the malformations of mild degree of my classification of the human cases; the hypoplasia of the tube and the absence of the epithelial anlage of the middle ear would, however, make it a malformation of severe degree. This discrepancy shows that a classification which is suitable to man is not always applicable to other species.

Rat E 6376, 15 days old. Mother received five injections before, one on the fifth and one on the 19th day of pregnancy.

The right auricle is reduced to a small hillock; no external canal is seen. The left auricle and external auditory meatus are normal. No other malformations are seen on the head. The brain shows internal hydrocephalus, otherwise there is normal configuration.

HISTOLOGICAL EXAMINATION OF THE TEMPORAL BONES (VERTICAL SERIAL SECTIONS)

Right side:

External ear. The auricular rudiment does not contain cartilage, but shows otherwise normal structure. The external meatus is completely absent and so is the tympanic bone.

Middle ear. The eustachian tube is well developed, the epithelial anlage of the middle ear is markedly hypoplastic. It extends for a short distance only backward under the cochlea. The middle ear space itself is also markedly hypoplastic; its main portion is filled with loose connective tissue. The space extends backward approximately to the level of the round window niche. The round window is present, the oval window missing. The epithelial anlage of the tympanic cavity is surrounded on its anterior, lateral and part of its inferior circumference by an irregularly cup-shaped bone (Fig. 7). The malleus, incus, stapes and tensor tympani and stapedius muscles are absent. The styloid is markedly hyperplastic and enters the middle ear space just behind and laterad to the bone just described (Fig. 8). One muscle bundle inserts on its medial and one on its lateral aspect. The former originates from the lateral wall of the labyrinthine capsule, the latter goes downward and joins the neck-muscles. The facial nerve enters the uppermost portion of the middle ear space, takes a lateral course along the roof and leaves the middle ear. The chorda tympani is absent. The geniculate ganglion is well developed. The stapedial artery enters the middle ear from below, behind the styloid, describes a quarter of a turn over its posterosuperior aspect in its course to the medial wall of the labyrinth (Fig. 8). It then goes forward and upward and eventually to the outer surface of the dura at a place much farther forward than normally.

The mandible and the temporomandibular joint are normal.

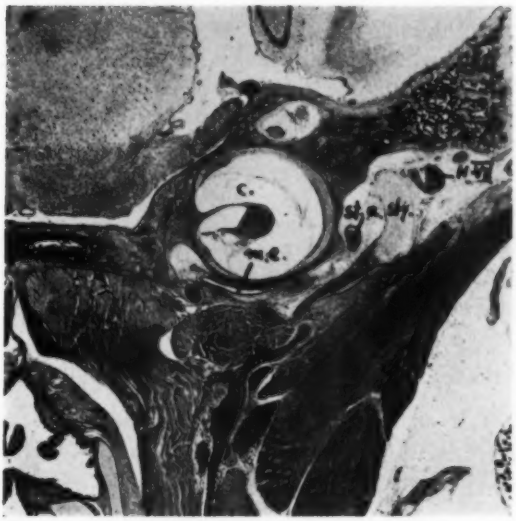

Fig. 8.—Rat E 6376. Right side. Section through the hyperplastic upper end of the styloid. The abbreviation c. indicates cochlea; m.e., epithelial anlage of middle ear; n.VII., facial nerve; st.a., stapedial artery and sty., styloid process.

Inner ear. The osseous labyrinthine capsule shows, with the exception of the absence of the oval window, normal configuration and structure. The membranous labyrinth is normal.

The left side shows no abnormalities.

Comment. Abnormal development of the posterior end of the first branchial arch caused the hypoplasia of the part of the auricle derived from the mesenchyme of this arch, absence of malleus, incus, the tensor tympani muscle and the tympanic bone; abnormal development of the posterior end of the second arch is responsible for the hypoplasia of the part of the auricle derived from the second arch, for the absence of stapes, oval window, stapedial muscle, the hyperplasia of the styloid process, the abnormal muscle bundles inserting on it, the abnormal course of the stapedial artery and probably also of the facial nerve through the tympanic space. Disturbances within

the first branchial cleft caused the defect of the external auditory meatus, within the first branchial pouch the hypoplasia of the epithelial anlage of the middle ear and of the tympanic space in general. Of particular morphologic interest is the cup-shaped bone which surrounds part of the epithelial anlage of the middle ear. It seems to represent a so-called rostral entotympanicum, a separate bone which forms in certain mammals the anterior portion of the floor of the middle ear[8,14,15,21] but has so far not been observed in any of the rodentia.

The case in question belongs to the malformations of medium degree with absence of the tympanic bone and hyperplasia of the upper end of the styloid.

Rat E 6378, 14 days old. The mother received one injection before, one on the sixth and one on the 20th day of pregnancy.

The right auricle is small and malformed; there is a small auricular appendage in front of it. The external auditory meatus is present. The left ear is normal. There are no other malformations on the head. The brain is normal.

HISTOLOGICAL EXAMINATION OF THE TEMPORAL BONES (VERTICAL SERIAL SECTIONS)

Right side:

External ear. The structure of the auricular rudiment is not unusual. The external meatus is well developed. The portion of the tympanicum which forms the wall of the external auditory meatus is slightly hypoplastic, the drum membrane is smaller on the left side. The tympanic bulla is present but markedly hypoplastic (Fig. 9).

Middle ear. The eustachian tube shows normal development and structure, the epithelial anlage of the middle ear, as well as the middle ear space itself, is markedly hypoplastic. The slit-shaped epithelial anlage extends for a distance backward under the cochlea; the remainder of the middle ear space is filled with mostly loose connective tissue. It extends backward to a point just behind the level of the oval window niche. The proximal part of the epithelial anlage of the middle ear adjacent to the eustachian tube is surrounded for a distance of less than 1 mm on its floor and its lateral side by a small piece of bone which in some places still contains areas of cartilage.

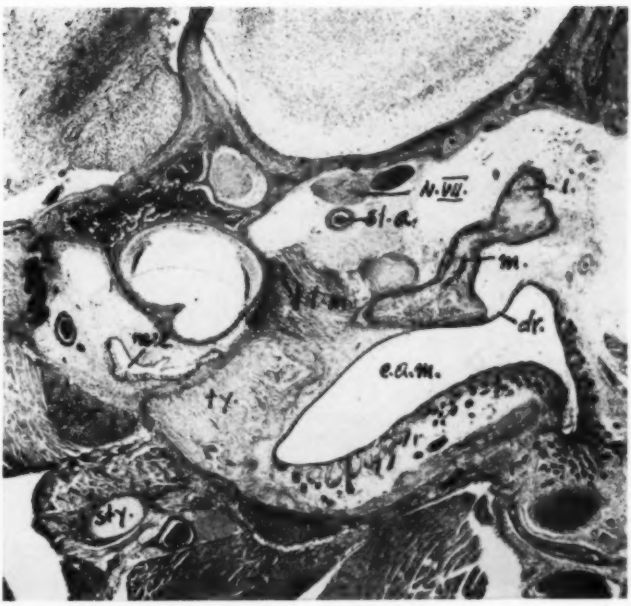

Fig. 9.—Rat E 6378. Right side. The abbreviation dr. indicates drum membrane; e.a.m., external auditory meatus; i., incus; m., malleus; m.e., epithelial anlage of middle ear; n.VII., facial nerve; st.a., stapedial artery; sty., styloid; t.t.m., tensor tympani muscle and ty., tympanic bone.

It is adjacent to but nowhere connected with the hypoplastic and malformed bulla which forms the main part of the bony floor of the middle ear space (Fig. 10).

On the medial wall, the round window is present but smaller than normal, the oval window is represented by a narrow fissure filled with fibrous tissue and lined on both sides with a narrow rim of cartilage. The malleus is relatively well developed, the tensor tympani muscle is present but short and plump. The incus is markedly hypoplastic, only part of the body being recognizable which articulates with the malleus (Fig. 9). The stapes and stapedial muscle are absent. The styloid is slightly hyperplastic; its upper end extends around the posterior margin of the hypoplastic bulla into the posterior part of the middle ear space.

The facial nerve goes through a short canal into the uppermost portion of the middle ear space. The geniculate ganglion is well developed. The nerve takes a straight laterad course and leaves the middle ear space between the squama and the posterior limb of the tympanic bone (Fig. 9). The chorda tympani is absent. The stapedial artery enters the middle ear space between the posterior limb of the tympanic bone and the cochlea, just behind the styloid process. It runs along the medial wall upward and forward (Fig. 9) and leaves the middle ear somewhat farther forward than on the other side, just in front of the cochlea, and ramifies as usual. The mandible and the temporomandibular joint are normal.

The inner ear shows no abnormalities, with the exception of the already mentioned abnormal configuration of the lateral wall of the labyrinthine capsule.

The left side shows no abnormalities.

Comment. Abnormal development of the posterior end of the first branchial arch caused hypoplasia of the part of the auricle derived from the mesenchyme of that arch, the anomalies of the tensor tympani muscle, the hypoplasia of the incus and of the tympanic bone; abnormal development of the second arch caused hypoplasia of the part of the auricle derived from the mesenchyme of that arch, the absence of stapes, oval window and stapedial muscle, the slight hyperplasia of the styloid process, the abnormal course of the stapedial artery and possibly also of the facial nerve through the tympanic space. Anomalies of the first branchial pouch led to the hypoplasia of the epithelial anlage of the middle ear and of the tympanic space itself. The hypoplasia of the drum is best explained by disturbances in the distal part of the meatal plate, a derivative of the first branchial cleft which forms the inner part of the external auditory meatus. The small piece of bone adjacent to the proximal portion of the epithelial anlage of the middle ear could again represent a so-called rostral entotympanicum which in this instance still shows its origin from a cartilaginous anlage.

The case just described belongs, in view of the fact that the external auditory meatus is present, to the malformations of mild degree of my previous classification.

Rat E 3, three days old. The mother received eight injections before, one on the first and one on the 15th day of pregnancy.

Fig. 10.—Rat 6378. Right side. Section through tympanic bone and rostral entotympanicum(?). The abbreviation e. indicates rostral entotympanicum(?); e.a.m., external auditory meatus; i., incus; m., malleus; m.e., epithelial anlage of middle ear and ty., tympanic bone.

The right auricle and the external auditory meatus are absent. The left external ear is normal. No other abnormalities are noted on the head. The brain is normal.

HISTOLOGICAL EXAMINATION OF THE TEMPORAL BONES (VERTICAL SERIAL SECTIONS)

Right side:

The external ear, meatus and tympanic bone are absent.

Middle ear. The eustachian tube is well developed and ends blindly. The epithelial anlage of the middle ear is missing. The middle ear space itself is short and narrow; it is filled with loose con-

nective tissue into which a slightly S-shaped rodlike piece of cartilage is embedded (Fig. 11). No other ossicles are seen. The tensor tympani and stapedius muscles and the styloid are absent. The stapedial artery and the facial nerve are not seen within the middle ear space.

Inner ear. The labyrinthine capsule is still cartilaginous with beginning ossification in some places. Definite windows are not recognizable although there is in one place a small gap in the lateral wall which is filled with connective tissue. The membranous semicircular canals, including the cristae, are normal. The subarcuate fossa is well developed. The utricle and saccule are very wide and communicate through a wide opening. The maculae are well recognizable. The endolymphatic duct and sac are absent. The cochlear duct is very wide, describes less than one turn and goes over directly into the saccule; the reunient duct is absent. An evaginated portion of the cochlear duct extends through the fundus of the internal auditory meatus backward into the subarachnoid space of the posterior cranial fossa (Fig. 12). It has the shape of a collapsed sac of 2 mm at its greatest length which overlies the posterior aspect of the petrous pyramid. The organ of Corti is reduced to a small mound of more or less undifferentiated cells; the stria vascularis is well developed, the cochlear portion thin, the spiral ganglion markedly hypoplastic. The facial nerve runs for a short distance in an anteromedial direction over the posterior aspect of the petrous pyramid and leaves the cranial cavity between the latter and the occipital bone. No trace is seen of the stapedial artery.

The mandible and the temporomandibular joint are normal.

The left side shows no anomalies.

Comment. Abnormal development of the posterior end of the first branchial arch accounts in part for the absence of the auricle, the presence of the rodlike ossicular rudiment which most probably represents the malleus, the absence of the tympanic bone and the tensor tympani muscle; abnormal development of the second arch accounts in part for the absence of the auricle, stapes, oval window, stapedial muscle, styloid, stapedial artery and the abnormal course of the facial nerve. Anomalies within the first branchial pouch caused the absence of the epithelial anlage of the middle ear and the hypoplasia of the middle ear space, within the first branchial cleft, the absence of the external auditory meatus.

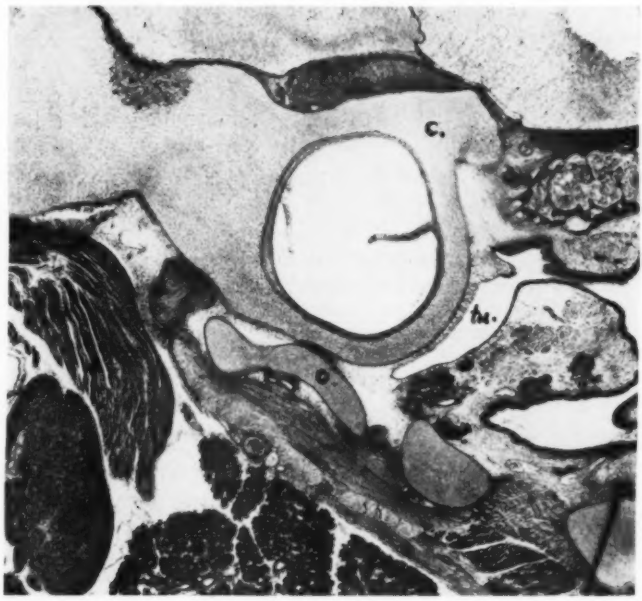

Fig. 11.—Rat E 3. Right side. The abbreviation c. indicates the labyrinthine capsule; o., ossicular rudiment and tu., Eustachian tube.

The malformation of the inner ear represents a relatively mild degree of the changes which were described by Hertwig[11] in the offspring of male mice which had received an x-ray dosage of 1000 r ("x-ray-mutated circler mice"). The changes are according to Hertwig the result of a primary displacement of the otic pit and vesicle which becomes separated from the neural tube by more mesenchyme than usual. This leads to disturbances of the inductory influences which emanate from the neural tube and which play a role in the normal differentiation of the otocyst. The changes found in rat D 9124 could be explained in the same way. The evagination of the cochlear duct through the internal auditory meatus into the subarachnoid space of the posterior fossa in rat E 3 was less marked than in Hertwig's animals. It is explained by her as a consequence of the increased endolymphatic pressure which forces the dilated cochlear duct through the area with the least resistance.

With regard to the malformations of the external and middle ear, the case just described can best be grouped with the malformations of severe degree of my previous classification.

Rat E 3550, two days old. Mother received eight injections before, one on the fifth and one on the 19th day of pregnancy.

The left auricle and external meatus are absent. The right ear is normal. No other anomalies are noted on the head. The brain is normal.

HISTOLOGICAL EXAMINATION OF THE TEMPORAL BONES (VERTICAL SERIAL SECTIONS)

No abnormalities were noted on the right side.

The auricle, external meatus and tympanic bone are absent on the left side.

Middle ear. The eustachian tube is short, the epithelial anlage of the middle ear is absent. The middle ear space is very narrow and filled with connective tissue. One ossicular rudiment is contained in the space. It resembles the malleus and is completely cartilaginous. It has the shape of a rod extending in a craniocaudal direction. Near its upper end a process resembling Meckel's cartilage is going forward and eventually joins the mandible. It is also cartilaginous but is covered on the lower part of its circumference by a thin shell of membranous bone. No other ossicles or any of the intratympanic muscles are visible. The styloid is absent. The facial nerve traverses the middle ear space just behind the ossicular rudiment. The stapedial artery goes upward and forward on the medial wall of the tympanic space and enters the cranial cavity at about the usual place. The chorda tympani is absent.

Inner ear. The labyrinthine capsule is cartilaginous with beginning ossification in some places. The round window is well developed, the place of the oval window is taken by a small fissure in the lateral wall. The membranous labyrinth is normal.

Comment. Abnormal development of the posterior end of the first branchial arch accounts in part for absence of the auricle, for the malformation of the malleus, of the tensor tympani muscle, the incus and the tympanic bone. Abnormal development of the second arch accounts in part for the absence of the auricle, for the absence of stapes, oval window, stapedial muscle and styloid, and for the abnormal

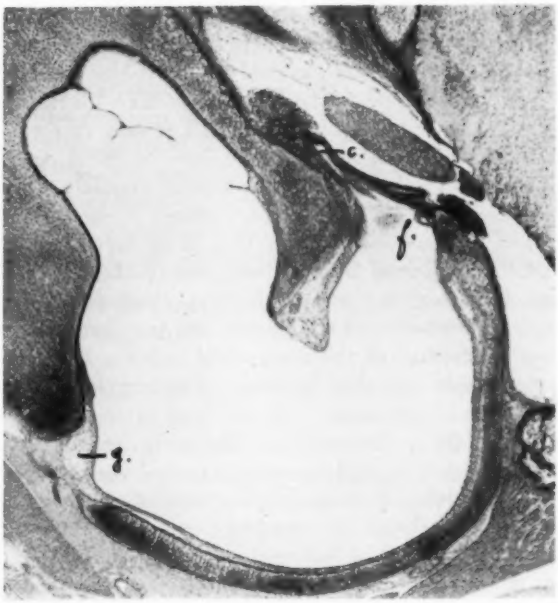

Fig. 12.—Rat E 3. Right side. Section through the fundus of the internal auditory meatus. The abbreviation c. indicates the evaginated portion of the cochlear duct; f., fundus of internal auditory meatus and g., gap in the external wall of the labyrinthine capsule.

course of the facial nerve through the middle ear space. The normal course of the stapedial artery seems noteworthy in view of the absence of the stapes and the other disturbances within the second arch. Anomalies of the first branchial pouch are responsible for the hypoplasia of the tube, the aplasia of the epithelial anlage of the middle ear and the hypoplasia of the tympanic space. The absence of the external auditory meatus is explained by lack of development of the primary meatus and the metal plate.

This case, too, can best be grouped with the malformations of severe degree of my previous classification.

COMMENT

A comparison of the findings in rats with those in all the reported human cases shows involvement of the same areas in both instances.

The stapes and the stapedial muscle were absent in all the rats, even in the malformations of mild and medium degree, and furthermore the styloid process was absent in three instances. This can be taken as an indication that in rats the derivatives of the second branchial arch are more susceptible to malformations than in man where absence of the stapes and the stapedial muscle is very rare and confined to atresias of severe degree. The hyperplasia of the upper end of the styloid in two cases indicates that this type of anomaly which is quite frequent in man (12 out of 63 observations) is also not uncommon in rats. The anomalous course of the facial nerve in all the cases is satisfactorily explained by the anomalies in the upper portion of the second branchial bar which shows a close developmental and topographic relationship to the nerve; the simultaneous absence of the chorda tympani in all the cases could indicate that this anomaly is more common in rats than in man. The complete absence of the epithelial anlage of the middle ear in three of the five cases makes it probable that this condition too is also more frequent in rats than in man and points to a greater susceptibility of the distal part of the first branchial pouch. Even when the epithelial anlage of the middle ear was completely absent, the tympanic space was present, although hypoplastic. This could be interpreted as evidence of a certain independence of the anlage of the tympanic space from the epithelial middle ear anlage. It deserves interest in view of the still disputed developmental mechanism of pneumatization.

Inner ear malformations, accompanying the malformations of the outer parts are evidently not less frequent in rats than in man. The type of inner ear malformation present in the two rats is, however, different from the types observed in man. It is noteworthy that in both instances the inner ear malformation was identical with those found by Hertwig in the offspring of male mice which had received irradiation. There are apparently many ways in which such malformations might be produced.

Again the right side was much more frequently affected than the left side (4:1), a fact (in man) which has already been pointed out by me and other authors.[11,12,18] Hertwig noticed the same greater susceptibility of the right side in malformations confined to the inner ear.

The fact that the temporomandibular joint was normal in all the cases, even when the derivatives of the upper end of Meckel's

cartilage were severely affected, is noteworthy. The effect of the abnormal developmental influences was evidently confined to a narrowly circumscribed area.

Further examinations in many more animals and in different species will be needed in order to confirm or refute the impression gained by me in analyzing the findings in the malformed rats. The way in which the malformations manifest themselves in a given area in different species seem to depend on inherited factors which are responsible for the greater or lesser susceptibility of certain structures to abnormal developmental influences whatever their nature might be.

An analysis of the genetic and the possible environmental factors which could produce congenital atresia of the ear as isolated malformation or in combination with malformation of other parts of the body was given in a previous paper;[1] nothing essentially new has been added since to our knowledge. Further discussion shall, therefore, be confined to the effects of trypan blue. Gillman and associates[9] found that the subcutaneous injection of one cc of an aqueous solution of trypan blue every two weeks into female rats before conception and during pregnancy led to a high incidence of such congenital malformations as hydrocephalus, spina bifida, eye defects, and a lesser incidence of tail defects, meningocele, harelip, cleft palate, cranioschisis, ear defects, imperforte anus, club feet and dislocations of the forelimbs and hindlimbs. If the animals received the injections on the seventh or sixth day before conception as well as on the seventh or eighth day of pregnancy, the incidence of malformation was 80 per cent and 65 per cent respectively.

Malformations of the ear were found in 5.9 per cent of the total number of abnormal animals. The malformed ears were either solitary anomalies or combined with others, such as hydrocephalus (two of the five cases described in this paper), defects of the eyes or both, or with defects of the skull.

The great number of congenital defects noted on the young born to the treated rats leaves, in Gillman's[9] mind, no doubt that the defects were causally related to the injections. In all the thousands of rats fed on a similar diet as the treated animals not one of the malformations was ever observed by him.

According to Gillman, trypan blue is absorbed by the albumin fraction of the plasma protein and produces a metabolic disorder in

the animals given the injections. The dye does not enter the fetus; so the malformations were evidently the result of metabolic disease in the mother. The fact that the highest incidence of malformations occurred when the mothers were given injections on the seventh or eighth day of pregnancy does, in Gillman's opinion, not necessarily mean that certain tissues of the embryo are mare vulnerable at certain developmental periods than at others. The supposed metabolic disorder could lead to disturbances in the implantation of the embryo into the uterine wall, which takes place at that particular time, and which is a complicated process necessitating various types of adjustments in the embryonic membranes and in the maternal tissues. It is much easier to relate the critical period of vulnerability of the rat embryo to the time of implantation than to the degree of growth and differentiation of those primordia which later undergo a perverted development. Changes in the metabolism of the chorionic villi at this early stage, for example, may profoundly modify the subsequent growth of the embryo and lead to malformations.

Gillman's experiments were repeated in mice by Waddington and Carter[24] and by Hamburgh.[10] Since some of the abnormalities which may be produced by treatment with trypan blue may be expected to simulate genetic effects ("phenocopies"), Waddington and Carter as well as Hamburgh found it desirable to carry out the experiments with strains of animals in which mutant genes of the relevant kind are absent. Mainly for this reason the authors worked with an inbred CBA strain of mice in which gross abnormalities (such as hydrocephalus, tail abnormalities, etc.) do no occur with any appreciable frequency. The animals were injected with 0.5 cc of a 1 per cent trypan blue solution seven days after fertilization.

The main effects of the dye were firstly a general inhibition of development, leading to abnormal closure of the neural tube, with the latter showing kinks and bends. The dye seemed, furthermore, to exert a more specific effect on the body fluids and the circulatory organs, causing a marked widening of the pericardial cavity and a considerable dilatation of the blood vessels, particularly in the head. The authors mention furthermore that in amphibian embryos trypan blue suppresses the differentiation of neural structures and of the notochord. They point out that the comparatively slight abnormalities of those organs seen in the mouse embryos may possibly not be only secondary consequences of mechanical disturbances but may in part be produced by a direct action of the dye. Ear changes are

not mentioned, except that otocephaly was observed in one single case.

Hamburgh injected females of the Bagg albino mouse strain with 0.5 cc of a 0.5-1.0 per cent trypan blue solution on the eighth day after fertilization. Among 571 embryos examined, 8½ to 14 days old, 332 or 58 per cent showed anomalies of two regularly occurring types: 1) Those of the anterior axis, such as brain malformations, resembling the condition known as pseudencephaly or exencephaly, and 2) those of the posterior axis, such as tail malformations resembling in some respects the phenotypes of certain tail mutations in mice ("phenocopies"). Minor effects on notochord and somites were observed too. Histological analysis showed the nervous system and the circulatory system to be most severely affected by the trypan blue treatment. Again, no mention is made of the condition of the ears.

"Essentially two alternatives present themselves for an explanation of the possible physiological mechanism of the dye: 1) The trypan blue at the dosage used might produce a subthreshold poison in the injected organism, which, unlike the dye itself, might be capable of passing through either the placenta or the yolk sac, or through both. In the embryo such a poison might exert physiological effects which in the growing and differentiating embryo might lead to the morphological consequences described. 2) The trypan blue molecules accumulating in the placental yolk-sac tissue might form an obstruction blocking the passage of vital substances to the embryo. Such a block might be of general nature or might be specific for a particular sustance."

The results of the two authors seem to confirm the contention of Gillman and associates that the malformations in rats were actually the result of trypan blue and not, as one might think, of incidental gene mutations in a genetically heterogeneous random group of animals. The significance of these investigations is quite obvious; they seem to open up a new approach to the problems of the causal genesis of malformations. It is to be hoped that eventually they will lead to a better understanding and possibly also to the prevention of certain malformations.

The relative significance of genetic and environmental factors in the etiology of congenital atresia remains still undecided.[1] The results of the histological examination of the ear region in animals in

which the malformations are undoubtedly of genetic nature would be essential to decide the important question of whether the histological findings in the two groups are identical or different from each other and whether the same or different mechanisms are responsible for their origin.

SUMMARY AND CONCLUSIONS

1. The results of the histological examination of the temporal bones are reported in a case of dysostosis mandibulofacialis[7] with bilateral atresia of the external auditory meatus. Both temporal bones belong to the malformations of medium degree with the atresia-plate formed mainly by the platelike tympanic bone. This brings the total number of usable histological observations to 63; 36 of them belong to the malformations of medium degree.

2. The heads of five young rats with unilateral ear malformations were examined in serial sections. The mothers of these animals had received subcutaneous injections of trypan blue before and after fertilization; it seems very probable that the malformations were the result of these injections.

3. A comparison of the findings in man and rats shows that they are essentially identical although in rats the distal portion of the first branchial pouch and the derivatives of the upper part of the second branchial arch seem more susceptible to malformations than in man.

4. In two of the rats malformations of the inner ear were observed which were identical with those described by Hertwig in the offspring of male mice treated with x-rays ("x-ray mutated circlers").

5. Examination of animals with ear malformations of undoubtedly genetic nature seems essential in order to decide the important question of whether the findings in them are identical with or different from those in animals in which the malformations were evidently experimentally produced ("phenocopies") and whether the same or different mechanisms are responsible for their origin.

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LXXVIII

LATE RESULTS OF FACIAL NERVE REPAIR

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Facial nerve reconstruction anatomically and physiologically was done first by Sterling Bunnell[1] and the results published in 1927. Encouraged by his work we did our first repair in 1929 and published the results in 1930 and 1931.[2,3] This work antedated that of Duel and Ballance who published in 1932.[4] Their fine research established nerve grafting of the facial nerve on a firm basis though, again, Bunnell had done a multigraft previously.

The results of grafts of other peripheral nerves are not good especially if near the proximal ends of the long ones. The facial grafts and repair are somewhat better because this is a very short motor nerve so there is less degenerated area for the axones to grow through, to their ultimate destinations.

The various types of intratemporal nerve repair are always accompanied by a "bony decompression" of the nerve in its course through the Fallopian canal. The extent of this so-called decompression varies according to the site of injury, i.e., it may be in the horizontal portion alone, in the vertical alone or in both. I realize that we do not see as many patients with surgical trauma now as in the pre-antibiotic days when simple mastoidectomy was more common. The common injury site in the radical mastoidectomy is in the horizontal portion or in the bend, while in the so-called simple operation the vertical portion just below the bend was most frequently affected.

A word on the so-called decompression: If pressure on the nerve from a depressed and impinging bone fragment is causal, then freeing the nerve from the constricting bone may suffice, but if it be a Bell's palsy or a partial injury with constricting bands of a fibrous nature, then bony decompression is not sufficient. A neurolysis or epineural

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Fig. 1.—Mrs. H. Nerve graft, early result. Fourteen months after operation.

sheath splitting along the long axis of the nerve must be done else regeneration fails. This point has not been sufficiently emphasized.

There are many reports on results a few years after operation but with one possible exception no reports of 18 to 26 year results. The question arose, "What could be the difference between two-year results and the later ones?" This is what I proposed to find out. It proved to be quite a task as some of the patients could not be found while others proved to be reluctant, and refused to answer any request for pictures or to answer questions. In the case of those who were minors when injured and still are, the inquiry was not pursued if their attitude was still that of great resentment toward the original operator.

Fig. 2.—Mrs. H. Nerve graft eighteen years after operation.

Six patients were contacted on whom repairs were done 18 to 26 years ago. The types of repair were end to end anastamosis, neurolysis and nerve grafts.

The time between injury and repair varied from twenty days to two years and the results of repair when done under one year were good. In one patient whose repair was two years after injury a permanent muscle elongation occurred which, of course, prevented good facial symmetry. Holmes and Young[5] found experimentally that Schwann cell activity is greatest two to three weeks after severance. Unions made after this time are more successful than the immediate union but, after 100 days there was less likelihood of success. Our experience does not so limit the chances of success.

Return of function varied from 30 days (neurolysis) to 7 months in nerve grafts. The rate of regeneration is given by Gutman, et al[6] as 3.42 mm with black silk suture of severed peroneal or tibial nerves in the rabbit. Plasma suture gave a figure of 3.53 mm. In the dog comparable methods showed 2.3 mm to 2.5 mm, while crushing injuries gave a rate of return of 4.36 + 0.24 mm per day. This was a measurement of the growth rate of enough fibers to give function and not that of single fibers.

The measurement was done by exposing the nerve in a given number of days and pinching it—going proximally until a reflex in the muscle was obtained. (All collateral blood vessel connections of the nerve are severed before the pinching experiments are begun.)

It is interesting to note that the elapsed time between injury and repair is the most important factor and that prepared grafts have shown no accelerating influence on function restoration.

No controlled experiments are possible in the human to determine the regeneration rate.

The form of questioning used, plus still photographs and one motion picture, has allowed us some tentative conclusions as to the late results of function return. For convenience the various facial nerve functions are separated and discussed more easily thereby.

Function return in three types of repair of the facial nerve was determined one to two years postoperatively and compared to the 18 to 26 year results. The results of this brief survey follow:

Tonus was good in all types of repair. This was the first function to return and was good early and late.

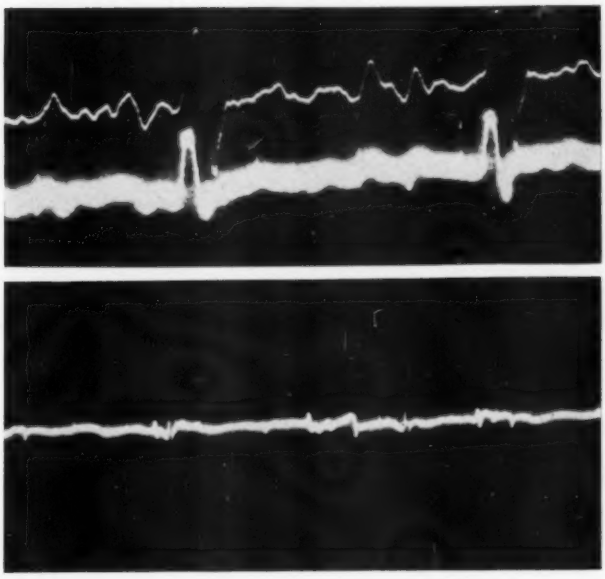

Fig. 3.—Mrs. C. Electromyograms; above, operated side, concentric needle electrodes in left orbicularis oris—3 mm apart. Shows synchrony and increased duration of motor unit action potentials; below, unoperated side, concentric needle electrode in right orbicularis oris. Shows normal action potentials.

Voluntary motor control was good in the first and second branches but absent in the frontalis.

Associated movements were least in the end-to-end anastamosis and neurolysis and more evident with the grafted nerves. They lessen through the passage of the years through conscious adaptation of the individuals to their difficulties of which they are painfully aware, or through a possible gradual return of the normal inhibiting mechanism.

There are many fascinating points regarding these associated movements.

Fig. 4.—Mrs. C. End to end anastamosis. One year after operation.

a) Are they due to nuclear changes or are they due to dichotomy of a descending axone with resultant rerouting to areas which that particular axone did not reach formerly?

We feel that Sullivan[7] and Wartenberg[8] and other proponents of the theory of nuclear change as the cause are correct. Dichotomy cannot explain the good return of pyramidal tract fibers with concomitant poor return of emotional control (extra-pyramidal) while the tonus (extra pyramidal) is good.

Wartenberg notes "that these associated movements occur in the occulomotor and facial nerve but not in other peripheral nerves.

"The definite nuclei and bundles are known in the oculomotor nerve and fibers from widely separated bundles would have to mix to explain the associated movements we see.

"One branch of the facial nerve may be injured and yet one gets the same associated movements as though the main trunk were involved, i.e., other branches which were not injured took part in the movements.

"The associated movements all follow a definite pattern which a hit or miss dichotomy could not explain.

"Histological evidence of nuclear change in the facial nerve after damage to the nerve has been known since 1893.

Fig. 5.—Mrs. C. End to end anastamosis. Twenty-six years after operation.

"A theory is propounded that this damage hits the more highly organized part of the nerve mechanism; leaving the phylogenetically older, tougher centers to return to their more primitive function. The newer centers which control orderly movements are damaged. This is the 'Release Hypothesis' of Hughlings Jackson used to explain similar effects in pyramidal tract lesions."

b) A second interesting point bearing on this arose on questioning a fine-looking twenty-year-old who apparently could not close his eye slit when asked. Fifteen years before he could do so (two years after nerve grafting) so we commented on the change. This intelligent lad then stated that he could not close the eye completely unless he relaxed and then concentrated on it. He said he had learned to avoid closing it tightly as the associated movements were marked and caused comment. He had established this protective mechanism so well that he had to will himself to close his eye tightly on command.

c) Experimenting on the residents and others convinced me that associated or mass movements are present in an uninjured patient on forceful attempts at movement, though to a lesser degree. One is able to close an eye without any associated movement but one cannot forcibly do so without the lower face showing an associated movement. The time between injury and repair had no bearing on the degree of associated movements.

Emotional control was poor in the early results—fair in the late results. The neurolysis and the end-to-end suture provided the best results—nerve grafting did not do as well.

Three of the nerve grafted patients showed poor or perhaps no emotional control, helping me to support my former statement that it never returned. The fourth patient's photographs of early and late return completely upset my former postion. The early history and photographs show no emotional control but in the 18-year picture you will note that it is good. (Figs. 1 and 2)

You will note further that this patient wrinkles her forehead better when smiling than when voluntarily attempting to. This curious phenomenon did not occur in any of the other patients.

Contractures have disappeared in all the patients studied for this report and in many others operated upon too recently to be reported here. Most of them disappear in two to five years.

Tics. Most of these fibrillary twitchings disappear after two to five years but some persist in the nerve grafted patients when they are mentally fatigued or emotionally disturbed. One football player noted that they never occurred when he was physically tired but occurred during college final examination time (at which time I happened to be checking him). The other types of surgical repair did not show these.

Tearing and frequency of winking are related. Tearing may occur when in a wind but it is never troublesome. Elapse of time showed no improvement in this.

Taste disturbances are not noted by the patients in any of the three types of repair. Tests of these were not made with the various substances usually used.

The findings just enumerated were observed and noted by me on examination of four patients. The three other patients who live a great distance from San Francisco submitted answers to a questionnaire and all of them submitted still pictures of various muscle actions as requested. The pictures of one of these latter were not clear enough to be of use though the questionnaire was intelligently filled out.

The reports of three patients follow to illustrate the late results in nerve grafting, end to end anastamosis and neurolysis.

Mrs. H. had a nerve graft done in 1937 following an injury to the horizontal portion of the nerve occurring during a radical mastoidectomy done elsewhere. Figure 1 shows her result 14 months after surgery. Figure 2 is the result after 18 years.

Mrs. C. had an end to end anastamosis done in 1929. The injury was a nerve severance just below the bend, caused by an operator searching too low for the antrum. The repair followed the injury by 1 year. Recovery was good in 14 months and has improved over the years.

Her electromyogram done 26 years after repair is shown in Figure 3. This was done by Doctor William Garoutte of the Department of Neurology of the University of California who reported, "The injured side showed the usual reaction of a lower motor neuron injury."

The response of the muscles to re-innervation are shown in 12 months in Figure 4 and in 26 years in Figure 5.

Fig. 6.—Miss G. Neurolysis. Eighteen years after operation.

Miss G. had a neurolysis done 142 days following a radical mastoidectomy (done by another surgeon) during which a portion of the nerve was scooped off resulting in a lateral neuroma and constricting fibrous bands. Figure 6 shows the result 18 years after corrective surgery, which consisted of trimming off the neuroma and slitting the fibrous bands transversely along the long axis of the nerve sheath.

CONCLUSIONS

Three types of intratemporal facial repair are presented with elapsed time from operation dates to time of preparation of present pictures and examinations from 18 years to 26 years.

The end to end anastamosis and neurolysis repairs show improvement over the years, though complete normality never occurs.

The nerve graft cases show little such improvement after the initial tonus and motor return noted at the one year postoperative period.

The main improvement factors were in lessened tics, disappearance of contractures, some return of emotional control and less marked associated movements.

Frontalis muscle function did not return.

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SCIENTIFIC PAPERS OF THE AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

(Continued from June issue)

LXXIX

ADDRESS OF THE PRESIDENT

CLYDE A. HEATLY

ROCHESTER, N. Y.

Members of the American Broncho-Esophagological Association and Guests:

It is my pleasure and privilege to welcome you to the thirty-sixth Annual Meeting of the Association. May I, first of all, express to you my deep appreciation of the distinguished honor which you have bestowed upon me. The opportunity of serving with the officers and council of your selection has been an experience which I will always cherish. I wish to thank each of them most sincerely for their interest and assistance.

I must confess at once that like Lot's wife, I have not been able to refrain from looking backwards. The penalty for me, however, has been profoundly satisfying. The progress of the Association, as charted so completely in its Transactions, has fully justified the faith and confidence of its founders. The liaison between our speciality and medicine has become increasingly close. We have moved steadily from the limbo of peripheral medicine toward its central and vital zone. The endoscopist has truly graduated from technician to consultant stature.

The development of broncho-esophagology has resulted from the tireless efforts of many individuals to increase the precision and accuracy of its techniques. The great advances made in thoracic surgery, anesthesiology and roentgenology have been complemented by many important contributions of the members of this Association. These include the recognition of the localizing value of the bronchopulmonary segments as well as an acceptable standardized terminology for their identification; important researches in pulmonary and esophageal physiology; improvements in the safety and scope of anesthetic agents; improvements in endoscopic instruments as well as visual aids to more accurate diagnosis; the development of improved methods of bronchography in both children and adults; the establishment of the diagnostic importance of the cytologic study of bronchial secretions and numerous other investigations of great value. Let us

not overlook, however, the tremendous contribution made by all of the members of this Association in teaching the value of bronchoesophagology throughout the land and in establishing by their individual efforts its permanent place in every medical center.

The life span of this Association coincides closely with the so called golden age of medicine — that fabulous period since 1910 which witnessed more progress in the control of infectious diseases than was made in all the years since Hippocrates. During this period the life expectancy in this country increased from 49 years to 68 years for men and to 71.5 years for women. The principal communicable diseases of childhood, which only a few decades ago had exacted such a heavy toll of child life, were reduced to vanishing proportions as a factor in morbidity. The death rate of pneumonia and influenza dropped from 202.2 per cent per 100,000 population in 1900 to 39.7 per cent in 1948, a decline of 81 per cent. The mortality rate in diabetes, despite an actual recent rise in the incidence of this diseases after the age of 45, decreased markedly reflecting in part the effectiveness of the control of the infections which had previously constituted such a hazard in its management. Tuberculosis, which at the turn of the century outranked every other cause of death, has now been relegated to tenth place and its toll reduced from 11 per cent to slightly over 1 per cent of all deaths. The recent closing of the world-famous Trudeau Sanatorium to patient care is a symbol of victory in one phase of tuberculosis control. The continuation of its laboratory facilities, however, emphasizes the continued need for vigilance particularly in view of the rising proportion of ambulant cases undergoing chemotherapy and the accompanying possibilities for reinfection as well as spread of the disease to other contacts. Thus the infectious diseases have been gradually replaced by the degenerative diseases as the principal causes of death in this country with heart disease first and cancer second in all surveys. Of particular interest is the sharp upward trend in the mortality from cancer of the lung which has increased during the past 35 years more than 20 times over previous periods. According to the statistics of the Metropolitan Life Insurance Company, the death rate from respiratory cancer in 1950-51 among insured white males between 45 and 75 years of age was 109 per 100,000 as compared with 72.1 for pulmonary tuberculosis and 50 for pneumonia. It is evident therefore that the entire pattern of disease in relation to both morbidity and mortality has undergone profound changes during the life of this Association.

The rapid development of the science of medicine in all its speciality fields has also created problems of first magnitude in med-

ical education. Many curriculum committees have attempted to solve the problem by increasing the total number of hours in the curriculum. Anderson, Manlove and Tipner found that in the period between 1948 and 1952 the average length of each academic year had been increased by one full week. A growing determined effort is now being made to refuse mere lengthening as a sound solution of the problem. George Packer Berry, the Dean of the Harvard Medical School, has stated that the "whole system of medical education urgently needs restructuring" and again that "it is essential that we regard with a critical eye the mounting tendency to impose specialization on the teaching of the medical student. I think," he continues, "it is tragic that the student is being confronted more and more by trees; has a chance to see the forest less and less clearly." The Commonwealth Fund in its annual report of 1950 states "If teaching is built around the need of a student who is learning as he grows and is not cut to a pattern determined by the competitive demands of medical school departments, it will gain coherence and pith." It is increasingly apparent that broncho-esophagology as well as its parent specialty, otolaryngology, must be alerting and preparing itself for these changing concepts of undergraduate education. Fletcher Woodward in his address to the Fifty-Seventh Annual Session of the American Academy of Opthalmology and Otolaryngology in 1952 emphasized that "the greatest criticism of Otolaryngology is its lack of interest in teaching at both the undergraduate and graduate levels. Its status today and its future lie in its awareness of this fact and its determination to correct it; and of these two duties, the most important is the teaching of the undergraduate student; for his knowledge of, his respect for, and his interest in otolaryngology are determined at this level." The need for responsible teaching is greater than ever before and broncho-esophagology must be prepared to do its full share in meeting this challenge. Now not only otolaryngologists, but internists, thoracic surgeons, gastroenterologists and anesthesiologists are interested in learning endoscopic techniques. The postgraduate teaching of broncho-esophagology, so magnificently begun by the first president of this Association, Dr. Chevalier Jackson, has been continued largely by otolaryngologists as part of approved residency training programs and in a half dozen medical centers in the form of short courses in approved methods. The constantly broadening interest in this field, however, has made existing educational facilities inadequate and there is great demand for short courses in the fundamentals of peroral endoscopy which are available to all qualified physicians. Broncho-esophagology adds a tremendous stimulation and value to any otolaryngological residency and the parent speciality must make every effort to continue the direction

and to broaden the availability of its teaching so that its years of leadership in this field are not lost. The reward will more than recompense the labor involved for there is no greater satisfaction than comes from a well developed training program.

In closing may I quote one of the great figures in contemporary medical education, Abraham Flexner: "Without ideals, without effort, without scholarship, without philosophical continuity, there is no such thing as education."

11 N. Goodman St.

LXXX

OBSERVATIONS ON THE HYPOPHARYNX AND CRICOPHARYNGEUS AREA:

A PLEA FOR GREATER ACCOMPLISHMENT IN ESOPHAGOLOGICAL PROCEDURES AND AN ILLUSTRATION OF TRANSESOPHAGOSCOPIC RETROGRADE DILATION

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In the following discussion I wish first to record some personal clinical observations and opinions relative to non-neoplastic conditions of the pharyngeal wall and musculature, especially that of the hypopharynx and cricopharyngeus area, with brief case reports to illustrate points under discussion; second to describe and illustrate a method of retrograde transesophageal dilatation that I have used for a number of years and which has been very effective and quite safe under antibiotic protection, and finally, to emphasize several points in the esophagological procedure which can lead to greater accomplishment.

The pharyngeal musculature is the dynamic factor in emptying the pharynx during swallowing, and is the true support of the pharyngeal wall. Much has been written about deglutition and I wish to avoid any detailed discussion but to emphasize observations of clinical importance on these structures.

First, a case is reported of dysphagia with a relaxed wide open cricopharyngeus, associated with unilateral X cranial nerve paralysis. I have observed relaxation of the cricopharyngeus to occur with both unilateral and bilateral X nerve paralysis in which the pharyngeal constrictor muscles were paralyzed on one or both sides of the pharynx. This case is presented to confirm the fact that the cricopharyngeus may be relaxed by interfering with the X nerve supply to this muscle, and to call attention to the fact that disturbance of the nerve supply to the cricopharyngeus may be useful in treating increased tone of this muscle. Also it emphasizes the fact that any

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portion of the hypopharyngeal wall that does not contract will decrease the efficiency of emptying the pharynx during swallowing.

CASE 1. A man, aged 62 years, entered the hospital on December 26, 1954, with the chief complaint of inability to swallow of four days' duration. He had felt well when he retired on December 22; when he awakened on December 23 his throat felt tight and he could not swallow solid foods. His condition became worse as the day progressed and he had difficulty with liquids which choked him, causing coughing, and some regurgitation. This condition persisted practically unchanged for two days. There was a past history of asthma and occasional choking spells. The patient said he had eaten chicken the day before his dysphagia began.

Fluoroscopic and roentgenologic examination had been made. The radiologist did not find organic disease but made a notation that there was no narrowing of the cricopharyngeus and that the patient had difficulty in initiating deglutition.

Esophagoscopy had been done and no abnormality had been noted to explain the patient's inability to swallow.

I was called in consultation on December 28, 1954. On looking at the x-ray (Fig. 1), it was evident to me that there was a left pharyngeal paralysis. Examination of the patient revealed a deviation of the palate and uvula to the right, mucus in the hypopharynx and marked weakness or paresis of the left vocal cord. Studies were carried out by the medical and neurological departments. The only significant finding was paralysis of the left tenth nerve. Swallowing had improved slightly by December 30, and the patient was discharged under the care of his physician. The diagnosis was paralysis of the left tenth cranial nerve, etiology unknown. It may have been the result of a vascular accident, localized neuritis, or a viral attack on the nucleus of the tenth nerve.

The roentgenograms (Fig. 1) and the outline drawings (Fig. 2*f*) demonstrate the typical findings of a unilateral left tenth nerve paralysis, that is, a relaxed open cricopharyngeus area and retained barium in the pyriform sinus and at the base of the tongue. Unilateral pharyngeal paralysis markedly interferes with deglutition. The pyriform sinus on the affected side does not empty. Mucus collects in the hypopharynx. Tracheal aspiration with coughing is the rule. Gravity alone, while helpful, is ineffective in emptying the pharynx on swallowing.

Patients with unilateral paralysis of the tenth cranial nerve, although inconvenienced by dysphagia and tracheal aspiration, may manage without help. Others, however, may require tube feeding to prevent tracheal aspiration and a tracheotomy for suction or aspiration of secretions from the tracheobronchial tree. With bilateral paralysis of the tenth nerve, frequently both tracheotomy and gastrostomy are necessary to protect the airway and properly nourish the patient.

Effective swallowing requires complete obliteration of the pharyngeal lumen. To accomplish this, the pharyngeal constrictor muscles of both sides must act in unison. During the contraction of the normal side, the paralyzed side bulges; its pyriform sinus remains full (Fig. 1); the desired mechanical effect is lost. Even though the cricopharyngeus offers no resistance, a portion of the secretions and food remains in the pharynx and pyriform sinus. Naffziger[5] has studied this problem and has given surgical aid. Any procedure such as plication, inversion or splinting that decreases the size of the paralyzed side of the pharynx and any muscle transplantation that elevates or constricts the pharynx should result in a greater mechanical advantage to the contraction of the constrictor of the opposite side, and thus improve swallowing.

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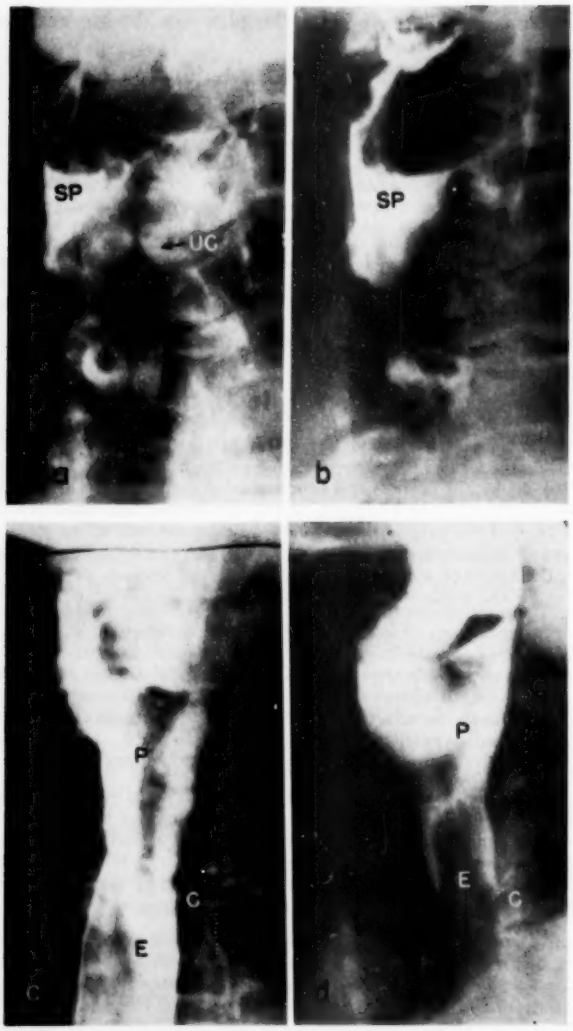

Fig. 1.—(Case 1). Pharynx and upper esophagus, using barium contrast media. *a*, Anteroposterior and *b*, lateral views showing barium-filled left pyriform sinus. *c*, Anteroposterior (see Fig. 2*f*), and *d*, lateral views showing hypopharynx continuous with the esophagus without constriction at the cricopharyngeus. S.P., Sinus pyriformis, left; V.C., Vocal cord; E., Esophagus; C., Cricopharyngeus, ordinarily closed, is relaxed and wide open; P., Pharynx.

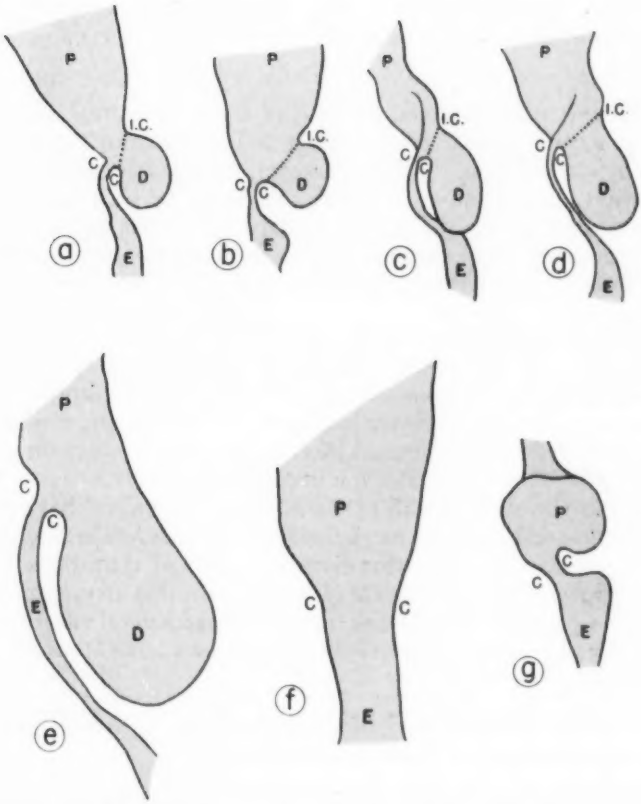

Fig. 2.—Outline tracings of roentgenologic studies of pharyngoesophageal area. *a*—*e*, Lateral views with diverticulum (D); *e* = Fig. 7*c*, Case 5; *f* = Fig. 1*c*, Case 1; *g* = Fig. 3*a*, Case 2 before tracheotomy. D., Diverticulum; P., Pharynx; E., Esophagus; C. C., Cricopharyngeus at pharyngoesophageal junction and the mouth of the esophagus; I. C., Notch made by margin of inferior constrictor muscle above mouth of diverticulum; Line C-I. C., mouth of diverticulum.

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There is an opportunity for members of this Society to improve upon the treatment of pharyngeal paralysis.

Secondly, in contrast to dysphagia due to paralysis and a relaxed cricopharyngeus constrictor, I wish to present a case to illustrate dysphagia caused by the constricting action of the cricopharyngeus muscles (Figs. 2*g* and 3) with good muscle tone of the pharyngeal constrictors, in which a diverticulum is not present. In fact, as yet no weak area has appeared and the pharyngeal wall has been equal to the pressure exerted upon it without herniation through the muscle. Here for the first time as far as I know is reported a case of local denervation of the cricopharyngeus with relaxation of this area for the relief of dysphagia, with entirely satisfactory relief of symptoms. It should also be noted that bilateral recurrent nerve paralysis does not produce relaxation of the cricopharyngeus.

CASE 2. A woman, aged 44 years, was referred to the Lahey Clinic July 26, 1954, with the diagnosis of a pulsion diverticulum which had been made by her local physician. The chief complaint was difficulty in swallowing of six months' duration, insidious in onset and becoming progressively worse. She also had difficulty in breathing following a thyroidectomy thirty-two years previously. Shortness of breath occurred on exertion and there was a loud snoring noise during sleep. She had been under medical observation and had taken desiccated thyroid the greater part of the last ten years.

The findings on physical examination were not unusual. Her weight was 132 pounds, the pulse rate 70, and blood pressure 122 mm systolic and 74 mm diastolic.

The laryngeal examination revealed bilateral paralysis of the recurrent laryngeal nerve, with laryngeal obstruction. The glottis was approximately 3 mm in width. Her shortness of breath was attributed to the bilateral paralysis and partial laryngeal obstruction of 32 years' duration.

A fluoroscopic examination was made by two radiologists and roentgenograms of the hypopharynx showed a dilated hypopharynx (Figs. 2*g* and 3*a*). Both radiologists called the dilated hypopharynx a pulsion diverticulum as did the referring physician, a diagnosis with which I disagreed.

A tracheotomy relieved her dyspnea. A Tucker valve tube was used. Later the pharynx and upper esophagus were explored through a lateral cervical incision as used for the operation upon diverticula. The surgeon did not find a diverticulum and dissected the cricopharyngeus area. The pharynx now empties completely on swallowing and no longer dilates. The dysphagia has been relieved.

In the next case there is just a suggestion of a diverticulum or slight dilation of the hypopharynx associated with a stricture at the cricopharyngeus.

CASE 3. A woman, 43 years of age, when first seen February 1, 1930, complained of dysphagia. She had been relieved by dilations in 1930 and 1931. She was treated for hypochromic anemia in 1933 and 1937. Anemia did not recur after 1937. Further dilations were carried out for dysphagia in 1937, 1942, 1944, 1945, 1946, 1948, 1949, 1950, 1951, 1952, 1953 and the last on January 15, 1955. The roentgenogram (Fig. 4) was taken January 14, 1955. Direct pharyngoscopy and dilation by the retrograde method illustrated using Jackson bougies and the 22 cm esophageal speculum have again given relief.

I believe a true diverticulum may be said to be present when herniation of the pharyngeal mucous membrane has occurred through the constrictor muscle. However, it may be difficult to determine when this occurs as muscle fibers may be pulled along with the mucous membrane and the transition from dilation of the pharynx to diverticulum may not be exact.

This case illustrates a beginning diverticulum which failed to develop probably because resistance at the cricopharyngeus was relieved by dilations (Fig. 4).

In Case 4 a small diverticulum did develop in the presence of a stricture of the cricopharyngeus. This patient had become markedly

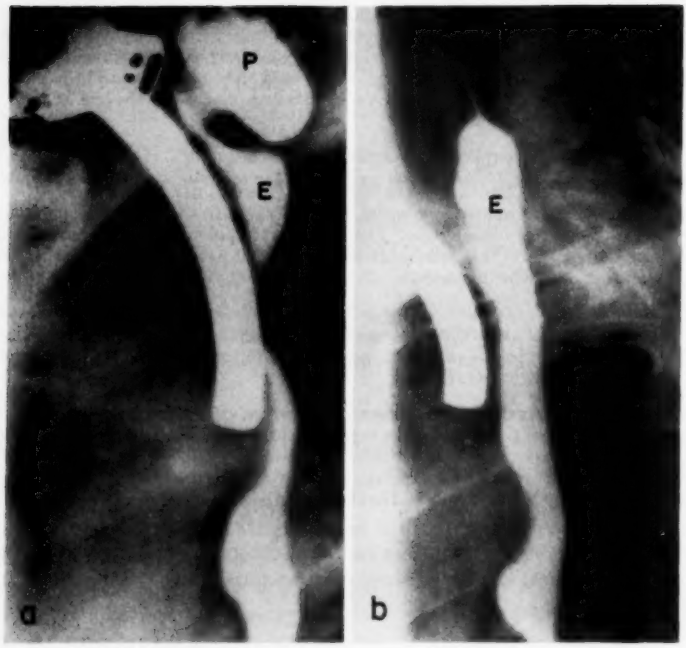

Fig. 3.—(Case 2). Hypopharynx and esophagus after a swallow of barium. *a*, Lateral view; P is the dilated hypopharynx. There is a constriction between the pharynx and the esophagus (see Fig. 2*g*). *b*, Lateral view postoperatively. The pharynx empties completely. The previous dilation, shown in *a*, cannot be demonstrated.

emaciated, losing one third of her weight. There was marked weakness of the pharyngeal constrictors. The surgeon and the internist with whom I saw this patient in consultation failed to realize the importance of the constriction at and of the cricopharyngeus even though this was pointed out to them. After operation and 21 months' treatment, dilation of the cricopharyngeus area gave marked relief in the presence of a recurrence of the diverticulum and severe dysphagia.

CASE 4. A woman, aged 75 years, was referred to the clinic on November 19, 1948, and complained that she was dying of starvation. She had been unable to eat an adequate amount of food for one year and six months and had lost 45 pounds, from 130 to 85 pounds. It was difficult for her to get food into the throat and to swallow it even after very soft food and liquids reached the hypopharynx. Solid foods could not be swallowed at all. There was no pain. In addition to dysphagia,

there was a history of glaucoma and a cataract extraction in one eye in previous years. There was no other pertinent history.

An examination revealed her to be very thin, a result of marked weight loss. Other than the eye there were no findings of significance. She was alert and intelligent. The blood pressure was 105 mm systolic and 68 mm diastolic, and the pulse rate 92. Laryngeal examination with a mirror showed good function of the vocal cords. There was a fair gag reflex and the palate rose to the posterior pharyngeal wall.

In attempting to swallow she held food in her mouth, having difficulty passing it from the mouth to the oral pharynx. Food and secretions remained in the pharynx for a considerable time after taking food and many attempts at swallowing were necessary before the pharynx was emptied of food. Saliva and mucus always remained in the hypopharynx.

Examination of the pharynx and esophagus using the barium swallow with fluoroscopic and x-ray exposures showed marked difficulty in the propulsion of the barium into the pharynx. After swallowing, there was retention of the barium in the vallecula and pyriform sinuses (Fig. 5*a*).

At the level of the seventh cervical vertebra there was a small, well-filled typical diverticulum measuring approximately 1.5 by 1 cm. It was the impression of the radiologist that in addition to the esophageal diverticulum there was a neurogenic disability of the swallowing mechanism.

On December 6, under direct hypopharyngoscopy, the hypopharynx was carefully visualized. There was no evidence of ulceration, new growth, inflammation or localized infiltration. The tissues were quite soft and pliable. The small diverticulum was readily visualized. I was unable to pass Jackson bougies through the opening on the anterior wall of the hypopharynx at the level of the cricopharyngeus even though I tried repeatedly. It was my opinion that there was fibrosis with stricture of the cricopharyngeus, and that the patient had a definite, marked muscular weakness of her pharyngeal constrictors without paralysis as the gag reflex was present and there was good vocal cord action.

I recommended a one stage excision of the diverticulum with repair of the pharyngeal wall and section of the cricopharyngeus muscle, to be followed by dilation.

This advice was not followed. Instead on December 13, the first stage of a two stage removal of the esophageal diverticulum was performed, dissecting the cricopharyngeal area and suturing the sac of the diverticulum upward. A Levin tube was passed into the stomach. Following operation, the patient was able to take fluids fairly freely but unable to take solid food. The second stage operation was never carried out. However, x-ray studies were made thirty-three days (Fig. 5*b*) and fifty-three days (Fig. 5*c*) after operation.

Following operation the patient was given intensive vitamin therapy and a high caloric, very soft diet, all foods being puréed. Although swallowing was improved, it never became normal. By March 24, 1949, she had gained to 92½ pounds and on May 23 weighed 98½ pounds. She was markedly improved. The patient remained under the care of her medical physician and was not heard from until September 21, 1950, when I received a telephone call stating that she had been unable to swallow for two days.

She was hospitalized and re-examined. She weighed 90 pounds. Roentgenologic and fluoroscopic examination of the esophagus revealed recurrence of the diverticulum (Fig. 5*d*) and marked narrowing at the cricopharyngeus. There was no evidence of growth. The cricopharyngeus was dilated up to number 45 French. There was definite resistance to the passage of the dilators. Cracking of the mucous membrane and bleeding were produced. She was placed on antibiotics. There was no spreading

Fig. 4.—(Case 3). Lateral view of very small diverticulum or dilation of the hypopharynx.

infection although the temperature rose slightly following this dilation. A week later a second dilation was done to number 45 French and she was again protected by antibiotics. She was discharged from the hospital improved October 4.

Her appetite was excellent. She could eat much better and was even taking solid foods. On October 19 she weighed 103 pounds and her weight increased to 112 pounds in November 1950. She has had no further difficulty in swallowing.

A series of outline drawings are presented of cases of diverticula to illustrate the changes that occur as the diverticula grow from small to large. The alteration in the outline of the hypopharynx and the dilation of the hypopharynx may be noted. Also, the changes that take place in the size of the mouth or the neck of the diverticulum and their relationship to each other and the opening of the esophagus through the cricopharyngeus can be seen. This change is illustrated in the outline drawings traced from roentgenograms of various diverticula (Figs. 2, 6, 7 and 8).

These outline drawings are tracings from lateral roentgenograms made during barium studies of the pharynx. The smallest diverticu-

lum was seen in 1952 (Fig 6*a*). The largest was treated in 1946 (Fig. 6*b*). The remainder were from patients seen during 1954. In the smallest diverticulum there is no distortion or dilation of the pharynx. The mouth and neck of the diverticulum are small. The mouth is in a vertical plane on the midline of the posterior pharyngeal wall. The neck protrudes at right angles to this wall posteriorly. The pharynx is funnel or V-shaped.

This small diverticulum in itself causes very little disturbance in the physical and mechanical emptying of the pharynx during swallowing. As the mucous membrane herniates through the inferior constrictor, the *mouth* of the diverticulum is indicated by a notch or a ridge made by the inferior constrictor (marked I. C. in the outline drawing, Fig. 2) at its upper and posterior limit and on the inferior anterior aspect as it passes over the cricopharyngeus (marked C on Fig. 2*a* to *e*). The line C - I. C. represents the plane and the anteroposterior diameter of the mouth. It is also true that the lateral dimension increases, so that the lateral dimension of the mouth of the diverticulum may become equal to the dimension of the pharynx. In the large diverticulum the notching of the inferior constrictor is no longer apparent on the posterior pharyngeal wall (Fig. 2*e*). The hypopharynx is dilated from V-shape to cylindrical. The mouth and neck of the diverticulum are continuous with it at its bottom. The opening into the esophagus shifts from a horizontal plane at the bottom of the pharynx to almost vertical plane on the anterior pharyngeal wall without an increase in size or apparent decrease in the tone of the cricopharyngeal constrictor.

With these changes there is a marked mechanical and physical disadvantage in the emptying of the pharynx. Food now enters the wide open mouth of the diverticulum at the dependent portion of the pharynx far more readily than at the small opening into the esophagus which is now on the anterior wall of the hypopharynx and held against the larynx by the cricopharyngeus muscle.

The diverticulum absorbs most of the thrust from the contraction of the pharyngeal constrictors as it drives the bolus of food downward during swallowing. The diverticulum may dilate under this pressure and a portion of the bolus escapes into the esophagus through the cricopharyngeus. As the constrictors relax there is a release of pressure in the diverticulum, the elastic, distended diverticular sac shrinks, and some of its contents are regurgitated into the hypopharynx.

The discussion and case reports given point out the basic changes anatomically and mechanically associated with diverticula.

Fig. 5.—(Case 4). *a*, Lateral view of diverticulum and filled pyriform sinus. *b*, Lateral view thirty-three days after operation. *c*, Lateral view 53 days after operation. *d*, Lateral view one year and nine months after operation.

PRINCIPLES OF REPAIR OF PULSION DIVERTICULA

Before discussing the treatment of diverticula, I would like to state briefly my personal view of their origin, for which I claim no priority or originality; it is that resistance to the passage of food from the pharynx through the cricopharyngeus into the upper esophagus, plus intrapharyngeal pressure, is the cause of a pulsion diverticulum. In other words, if there were a free passage of a bolus of food from the pharynx into the esophagus, there would be no excessive pressure in the pharynx to produce a herniation (diverticulum) through its wall.

In some cases I feel certain that resistance to the passage of food may be due to a fibrosis, stricture or web formation which occurs in the mucous membrane, submucosa and adjacent muscle and is an occasional etiological factor in producing diverticula. Also, there may be resistance to the passage of food through this area without true organic stricture, as just mentioned. Theories have been advanced to explain this phenomenon, which include increased tension or tone of the cricopharyngeus muscle, spasm of the cricopharyngeus muscle and inco-ordination in the relaxation of this muscle during the act of deglutition. The force of the pharyngeal constrictor muscles and associated muscles of deglutition working against this resistance creates sufficient pressure to produce a herniation in the weaker area of the hypopharynx which is consistently in the midline just above the cricopharyngeus. The support to the mucous membrane of the hypopharynx at this area of weakness is given by the inferior constrictor muscles which lie anterior to the vertebrae. This support may be decreased during the act of swallowing when the pharynx is pulled upward and forward away from the vertebral column.

I also believe that pulsion diverticula of the lower end of the esophagus would not occur if considerable pressure were not required to force the food from the esophagus into the stomach. This diverticulum also occurs in a weak area of the esophageal wall, but in such instances there is not a constant location of the weak point.

The surgical repair of pharyngeal diverticula should: (1) Remove if possible the etiological factor producing the lesion; (2) remove the diverticulum; (3) restore pharyngeal continuity and function.

I have recommended partial section of the circular cricopharyngeal fibers at the upper end of the esophagus in the angle between the esophagus and the diverticulum to decrease muscular tension and permit relaxation of this area, this being comparable to the procedure practiced in pylorospasm and cardiospasm. However, I recognized

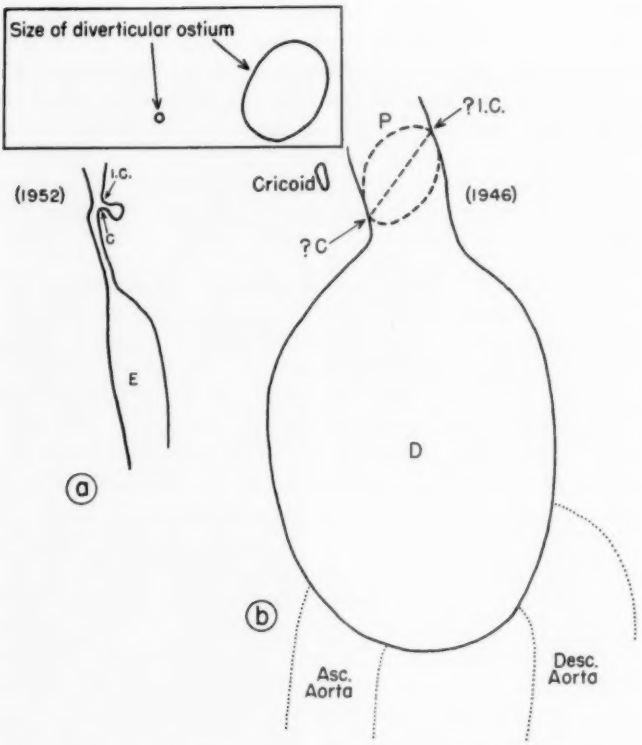

Fig. 6.—Outline tracing from roentgenograms, showing relative size of the large and small diverticula, relative size of the ostia, and the dilation that takes place in the hypopharynx.

that many cases were successfully treated without this aid, and I was unable to give a satisfactory explanation until the early 1930's when I observed the marked relaxation that occurs in the cricopharyngeus with X nerve paralysis (Fig. 1). The small terminal fibers to the cricopharyngeus may be stretched and pressed laterally as the mouth and neck of the diverticulum increase in size.

Many surgeons during the surgical repair of pharyngeal diverticula give little thought to weakening the cricopharyngeal muscle and do not make a deliberate attempt at the time of operation to weaken or relax this muscle. This end, however, may be accomplished without purposeful effort as careful dissection of this region, I believe,

interferes with its innervation, and hence, a weakening by paralysis or partial paresis of this muscle takes place, as occurred in Case 2.

Kaplan[3] has treated dysphagia due to paralysis (or marked weakness) of the pharynx following poliomyelitis by section of the cricopharyngeus, with improvement in deglutition. On occasion, I have also sectioned the cricopharyngeus.

In the removal of the diverticulum the mucosal sac should be dissected free from the musculature of the pharyngeal wall and excised, with repair of the opening in the mucosa followed by repair of the musculature opening.

In a small diverticulum the mucosal sac may be inverted without its removal and is safer than a two stage operation. So far as I know there is no contraindication to this procedure for small diverticula. Coburn[1] has recently reported the one stage infolding method of surgical treatment of diverticula.

The surgeon should restore the funnel shape to the hypopharynx with the opening of the funnel directly into the cricopharyngeus. This may require tailoring the hypopharynx especially in the larger diverticula, and shortening the distance between the inferior constrictor and cricopharyngeus in the midline posteriorly (line C - I. C. in Figure 7*c* and *d*).

The next case is one in which the surgeon failed to obtain a good result, and his mistakes are analyzed to emphasize the principles mentioned.

Case 5. This man, aged 44 years, gave a typical history and his roentgenograms were entirely characteristic of a true, large pulsion diverticulum (Figs. 2*e* and 7). The neck of the diverticular sac and the hypopharynx appeared particularly large and the hypopharynx itself was dilated.

The diverticulum had been removed by a two stage procedure. The patient accidentally pulled out the Levin tube soon after his second stage operation and it was not replaced immediately, during which time food escaped into the wound. The tube was replaced, the wound healed quite well and the patient returned home. A month later, at his home, a swelling and inflammatory reaction suddenly appeared in the neck. The following day there was difficulty in swallowing and on the third day the wound discharged, which was followed by the formation of a fistula.

The patient was seen in consultation. A Levin tube was inserted and the hypopharynx and upper esophagus were dilated. Outline drawings (Fig. 7) taken from roentgenograms revealed a dilation of the hypopharynx and a fistula. A portion of the diverticulum still remained. Apparently, at the first and second stage operation, an adequate amount of the sac had not been removed and the hypopharynx had not been infolded to decrease its size.

Operative repair was recommended. The fistula was closed and inverted, and the dilated hypopharynx was infolded without resecting any additional mucosa

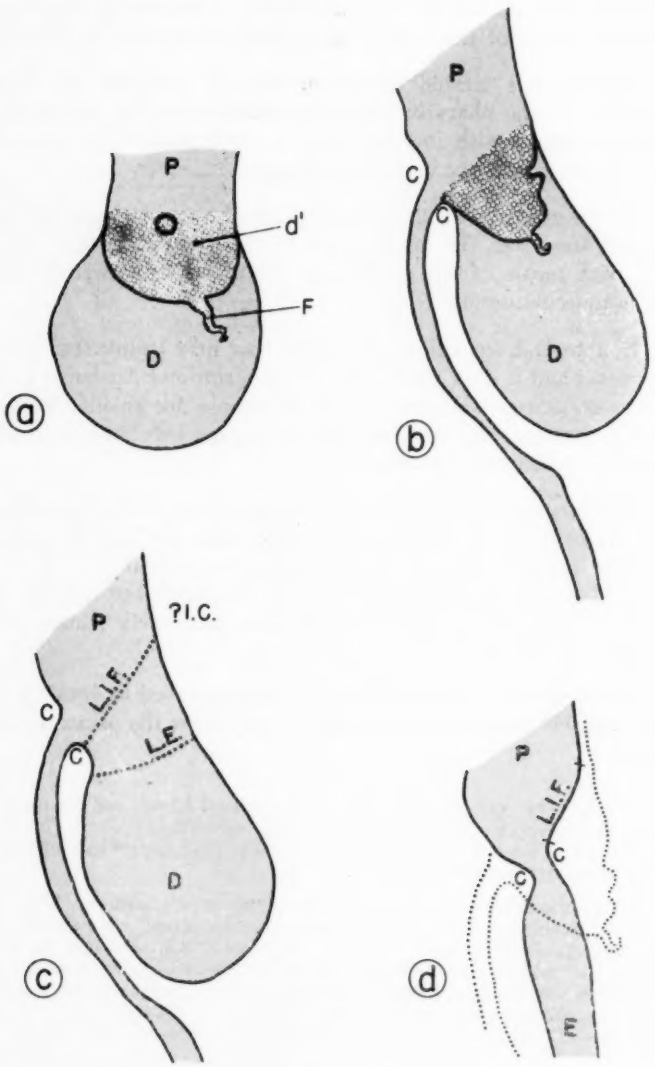

Fig. 7.—*a, b, c.* Outline tracings of roentgenologic studies of Case 5. P, Pharynx; D, Diverticulum; E, Esophagus; C C, Cricopharyngeus.

a, O, mouth of esophagus; d', residual diverticulum superimposed over D, original diverticulum, F, fistula.

b, Lateral view of residual diverticulum superimposed over original diverticulum.

c, Estimated line of excision (L.E.) at first operation; recommended line of infolding (L.I.F.) for repair; ? I.C., no roentgenologic evidence of exact border of inferior constrictor muscle.

d, Recommended repair with line L.I.F. shortened and the fistula and pharyngeal wall infolded into a funnel shape with the opening of the funnel at the cricopharyngeus (C C).

(Fig. 7*a* to *d*). The infolding extended for a distance of approximately 1 inch, tailoring the hypopharynx to a funnel shape. Convalescence was satisfactory.

A follow-up roentgenogram revealed the fleck of barium at the level of the cricopharyngeus and almost complete emptying of the hypopharynx on swallowing. A good functional result was obtained.

This case illustrates the marked dilation of a hypopharynx with the very large mouth and "neck" of the diverticular sac. It illustrates the formation of a fistula, a possible complication of not using a Levin tube, or of removing it too soon, and the lack of weakening of the cricopharyngeus muscle.

In this case the surgeon failed to decrease the dilation of a hypopharynx, leaving it rounded and bag-like (Fig. 7*a* and *b*), with the esophageal opening eccentrically placed. The mechanical disadvantage in emptying the pharynx has not been corrected. Secondly, he failed to make a muscle repair of the area through which herniation took place. When a muscle repair is not made that portion of the pharynx fails to constrict during contraction of the pharyngeal constrictor muscles, and the pharynx fails to empty completely. For this reason I have insisted upon infolding the lower portion of the hypopharynx in the midline or side posteriorly, suturing the muscles over the opening of the herniation. This lends muscular support to the mucosa and permits contraction of the hypopharynx in this area. This will decrease the size of the dilated hypopharynx, making it funnel-shaped (Fig. 7) while shortening the line C - I. C. brings the cricopharyngeus back in line at the bottom of the pharynx. Tailoring the hypopharynx to a funnel shape is more accurately and readily accomplished if the area has not recently been disturbed surgically. It is also important that the opening of this funnel be directly through the cricopharyngeus. The infolding procedure has been mentioned in relation to small diverticula and in the repair of the hypopharynx in large diverticula. On several occasions I have noted the hypopharynx to remain dilated after an esophageal diverticulum has been removed (Fig. 8).

When muscular repair of the pharyngeal wall is imperfect, this portion of the pharyngeal wall does not contract sufficiently to obliterate its lumen. In such cases secretions accumulate in the hypopharynx, and the hypopharynx fails to empty completely during the act of swallowing even though a true diverticulum no longer exists.

Case 6. This patient was seen by me in consultation December 15, 1954. However, she first came to the clinic in May 1952 with a typical pulsion diverticulum (Fig. 8*a*) and also a complaint of epigastric pain and distress. The diverticulum was treated surgically with a Lahey two stage operation, with relief of the gurgling

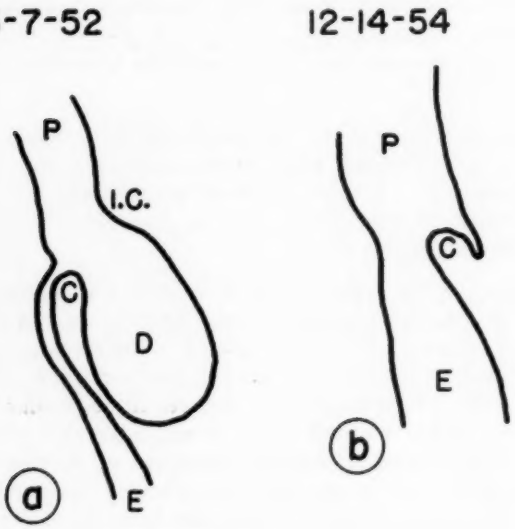

Fig. 8.—(Case 6). *a*, Line drawing of roentgenogram; lateral view of pharynx taken in May 1952, showing typical diverticulum. *b*, Two years and seven months (taken in December 1954) after surgical repair, showing relaxed cricopharyngeus and dilated hypopharynx.

and a constant desire to swallow. Her epigastric distress was treated symptomatically, with marked improvement but never complete remission. During the six months before I saw her, the epigastric pain and distress had increased, and quite severe pain at times extended into the right side. Tracings from a roentgenogram (Fig. 8*b*) showed that the diverticulum had been removed, the cricopharyngeus was relaxed and the pharynx has remained dilated. The posterior pharyngeal wall had not been inverted or the muscular wall repaired, yet clinically the result was entirely satisfactory. On review of her previous roentgenogram and on complete examination in December 1954 it was observed that this patient had also a hiatal diaphragmatic hernia, a slightly shortened esophagus and esophagitis which were the causes of epigastric distress and chest pains. They have now been relieved by appropriate treatment.

This case illustrates (1) the presence of multiple esophageal lesions; (2) the value of complete esophageal examination; (3) the fact that excellent clinical results may follow imperfect technical procedures (Fig. 8*b*). However, one should strive for technical perfection, which, if attained, leaves less to chance.

In cases in which dysphagia remains after the diverticulum has been removed, dilation of the upper esophagus and cricopharyngeus area may be given a trial with the hope of improvement, as in Case 4.

Dilations of the cricopharyngeus may decrease the resistance to the passage of the bolus of food through it and be beneficial to patients with small diverticula, to those unfit physically for operation and to those who refuse surgery.

Although dilations may be quite helpful in some instances, they are unsatisfactory in that they do not remove the diverticulum or repair the opening through the constrictor muscle. It may be necessary to repeat dilations from time to time.

Although many patients may do well without a Levin feeding tube following excision of a diverticulum and some surgeons advise against its use, I would use it routinely for a minimum of seven days in any case in which the pharyngeal wall has been opened; the larger the defect the more important its use. If for any reason the Levin tube is removed, then intravenous feeding should be used for a similar period.

Should there be evidence of delayed healing, a longer interval of rest for the pharynx is indicated.

Since 1928 I have had the privilege of being associated with the late Dr. Lahey who had an intense interest in and a vast experience with pulsion diverticula. Since 1923 Dr. Lahey has given numerous discussions and addresses on pulsion diverticula and has written twenty papers on this subject. His most recent summary[4] and convictions were published with Dr. Warren in January 1954, in which some 365 operated cases were reported up to May 1953.

I am in agreement with much of this article. My interpretation of cause and effect varies somewhat from Dr. Lahey's. However, his technique when fully carried out took care of most of the discrepancies and he obtained excellent results.

Dr. Lahey was a firm believer in the two stage procedure, and thought that small diverticula should be left until the sac of the diverticulum developed to such a size as to make it easy to find and dissect the neck and main portion of the sac to insure safe removal at a second stage. On the contrary, I recommend a one stage operation for small diverticula. Since most diverticula progressively increase in size and since the presence of a diverticulum is a possible menace to the health of the individual, its surgical repair should not be unduly delayed after the diagnosis has been made. From the previous discussion in this paper it is apparent that even the smallest diverticulum may be repaired and I believe should be unless the condition of the patient contraindicates surgical intervention or life

expectancy is short. The small diverticulum, of course, is not a surgical emergency and operation can be carried out at the convenience of the patient.

To me it is apparent that there is a great difference in the problem of repair of the very small diverticulum and the very large intrathoracic diverticulum. It is in the latter that I would recommend the Lahey two stage operation. If the neck of the diverticulum is carefully dissected at the first stage (a point insisted upon by Dr. Lahey), the muscle repair and the tailoring of the hypopharynx to a funnel shape should be done at that time as at the second stage the tissues are thickened by swelling and are friable, making identification and suture more difficult.

An alternative in the two stage procedure for the large diverticulum is to dissect free only the lower mediastinal portion at the first stage, and allow the mediastinum to wall off. In the second stage the neck of the diverticulum and the pharynx are dissected, the sac is removed and the pharynx repaired as advocated in regions not previously disturbed by operation.

TRANSESOPHAGOSCOPIC RETROGRADE DILATION

The successful use of this method of esophageal dilation for a number of years has convinced me that it deserves description and further publicity. No new or unusual instruments are required, only an esophagoscope or speculum and a set of Jackson dilators.

Retrograde dilation is begun after the largest Jackson dilator that can be passed through the esophagoscope has been used. Two smaller dilators are passed one at a time through the esophagoscope and through the stricture to a position below the stricture. The two dilators are then pulled up simultaneously into the stricture from below, producing further dilation. Many combinations are possible; three, four and even five dilators may be passed one at a time and brought simultaneously into the stricture from below. An illustration of some of these combinations can be found in the Surgical Clinics of North America, June, 1955, page 632.

When multiple dilators are used, the largest is passed first. Multiple dilators may be removed trailing after the esophagoscope as it is removed.

When dilation is so forceful that the mucous membrane or the esophageal wall is cracked and bleeds, antibiotics are given prophylactically to prevent the spread of infection.

CASE 7. A woman, aged 28 years, was referred to the Lahey Clinic on November 3, 1954, for possible surgical treatment of a stricture of the esophagus which had been under observation and treatment for thirteen years. The symptoms on admission were dysphagia (only liquids could be swallowed), loss of weight, epigastric and substernal pain and burning which became more severe when she was lying down.

Her trouble began more than thirteen years ago following a severe and protracted episode of vomiting. For the past eleven years she had been treated by a competent esophagologist. His diagnosis was congenital short esophagus with ulceration and cicatricial stenosis at the esophagogastric junction.

The general examination revealed that the patient was thin; she weighed 95 pounds. All other physical, laboratory and roentgenologic examinations were negative except the roentgenograms of the chest which revealed infiltration of the left upper lung and some mediastinal adhesions consistent with chronic pneumonitis probably caused by aspiration. Fluoroscopy and roentgenographic examination of the esophagus revealed a moderately dilated, short esophagus, a narrow stricture at the esophagogastric junction and a diaphragmatic hernia. Esophagoscopy was carried out under general anesthesia and an esophagitis at the lower 1 cm of the esophagus with granulation in the stricture was found. Transesophageal retrograde dilations were carried out as described. The esophagoscope was passed through the stricture into the stomach. The stricture was approximately 2 cm long. At the time of the esophagoscopy and dilation, antibiotics were given to protect against the possible spread of infection. Following esophagoscopy, web bougies were advised for self-dilation. Since her discharge from the hospital five dilations have been made over a previously swallowed thread, an office procedure. A number 42 French bougie is now being passed by the patient. She is receiving a modified ulcer diet, antacids, and sleeps with the chest elevated. Dysphagia has been relieved and she seldom has epigastric discomfort.

This case illustrates the result of aggressive dilation. Since esophageal dilation and instrumentation may be dangerous, the degree and rate must be governed by the judgment of the esophagologist.

I have found this procedure especially effective in single short strictures even though they are of long duration and heavy scarring has taken place. If this method is adopted one should seldom hear, "We passed the largest size dilator that would pass through the esophagoscope and could not do more." More can be and often should be done. It should be emphasized that this is not the only method of dilation but it has often enabled me to accomplish satisfactory dilation when other methods have not been feasible during esophagoscopy.

The thoracic surgeon and others, who might call us as consultants, are performing endoscopic procedures themselves for two reasons. The first is as a matter of convenience and the second is that they are dissatisfied with the information obtained by consultation.

If we can give outstanding service and performance (which we should be able to do) well beyond their capability, the balance might be tipped in our favor.

Over the years I have made my quota of mistakes and with the hope of preventing their repetition, I am emphasizing some facts and procedures that have led to greater accomplishment in my work.

The esophagologist is one of a team for the study of the patient and he should be very willing to assume his responsibilities. He must always bear in mind that esophageal disease and symptoms are frequently associated with generalized or systemic disease. A detailed history and complete examination are always in order. Besides obstructive symptoms, any vague symptom in the neck, chest or upper abdomen increasing to and including excruciating pain can be produced by esophageal disease.

The esophagologist who accepts a history without personally quizzing the patient and reviewing the history with him will miss an opportunity to improve his work.

The fluoroscopic and roentgenologic examination is extremely important and should not be neglected even though it is not conclusive. To be of greatest value it should be made in the presence of the esophagologist who acquaints the radiologist with the symptoms of the patient, the possible type and the location of the lesion. Contrast media are selected that are suitable for the particular case and the patient is placed in the position that will give most information concerning the esophageal condition.

I have caused injury to the cervical esophagus with mediastinitis resulting, by pressure of the esophagoscope against exostoses and spurs of the cervical vertebrae during lower esophageal and gastric examinations. I make routine lateral cervical roentgenograms to be forewarned of their presence so that injury may be avoided. Injury to the esophagus and spreading infection from trauma are avoided by using muscle relaxing general anesthesia and antibiotic protection.

In the presence of a large diverticulum or a dilated esophagus these cavities are emptied and cleansed by lavage as one does other portions of the gastrointestinal tract before endoscopic study. Too frequently I have seen the esophagoscope passed into an esophagus filled with debris.

Local anesthesia was used in my early training but frequently left much to be desired. Association with anesthetists capable of intratracheal intubation with complete control over respiratory function and with the use of muscle relaxing drugs is most helpful. Such an anesthesia eliminates muscle tension and straining, rendering the endoscopic procedure more accurate, easier to perform and less hazardous.

During esophagoscopy visualization is greatly enhanced by distending the lumen of the esophagus by gentle air pressure using a lens to close the proximal end of the esophagoscope. This is especially

helpful in the large or dilated esophagus, in visualizing the stomach wall, gastrojejunal stoma and on occasion the intestinal loops. Inflation has helped in the evaluation of the fixation or flexibility of the walls of the viscus and their texture. It has helped in evaluating strictures and in passing instruments through narrow or tortuous areas and in passing the esophagoscope from the esophagus into the stomach in cases of cardiospasm. On occasion, change of position of the patient, as elevation of the chest with pressure upon the abdomen, may help in visualization of the gastric walls.

Occasionally, the previously swallowed thread has been a great aid in passing a feeding tube over the thread and in passing the esophagoscope, using the thread as a guide in both pulsion diverticula and cardiospasm.

Since the purposes of esophagoscopy are both diagnostic and therapeutic, one should exploit the opportunity to accomplish both purposes. The esophagologist must not be satisfied too easily. The examination should be as complete as possible, compatible with safety. The stricture should be dilated as previously described, using the Tucker-Plummer dilator in cases of cardiospasm. It should be remembered that multiple lesions are common in the esophagus. The possibility of cancer should never be forgotten. Biopsies should be taken from characteristic areas of the disease. The esophagologist should never be complacent if the biopsy report is negative. A negative cell study acts as a narcotic that dulls diagnostic endeavor. In cases of suspected malignancy of the upper two thirds of the esophagus and mediastinum, bronchoscopy as well as esophagoscopy should be done.

Such aggressive endeavor may be considered dangerous, yet I have not found it so, especially when appropriate and immediate antibiotic protection is available and is used when trauma is produced, as in instances of forceful dilation, in deep biopsies, or when sharp cervical spurs are present. During the past eight years at the Lahey Clinic mediastinal drainage has not been necessary to treat mediastinitis produced by endoscopic procedures.

Although the above discussion is incomplete and nothing new has been mentioned, these points deserve repetition as they may lead to better performance by us, better service to our consultants and greater benefits to our patients.

SUMMARY AND CONCLUSIONS

I have attempted in this paper to point out some basic facts, observations and methods which in my own experience have made

greater accomplishment possible. The history and physical condition of the patient should never be neglected. Special attention is directed to the roentgenological examination of the pharynx and esophagus; the roentgenological examination of the cervical spine for exostoses before esophagoscopy; the preparation of the esophagus by lavage; the use of general anesthesia with muscle relaxation and tracheal intubation; the inflation of the esophagus during examination; the possibility of multiple lesions in the esophagus; the use of antibiotics to prevent infection following trauma, and the fact that the negative biopsy is no excuse for complacency.

A method of retrograde dilation of the esophagus through the esophagoscope has been presented and an illustrative case reported.

Vagus or X cranial nerve paralysis is discussed and an illustrative case reported. Attention is called to the resulting relaxation of the cricopharyngeus which remains open; also that the pharynx is not completely emptied without complete contraction of the pharyngeal wall. These are basic considerations in surgical treatment of dysphagia, due to X nerve paralysis, and emphasize the importance of repair of the muscular pharyngeal wall and the infolding of the hypopharynx to correct the dilation that occurs with large diverticula and that the cricopharyngeus can be relaxed by interrupting its nerve supply.

Dysphagia due to cricopharyngeal spasm or hypertonicity is a clinical entity (Case 2) that can be relieved by local interference with the X nerve supply to this muscle by carefully dissecting this region.

The cause of increased tone, spasm or inco-ordination of relaxation of the cricopharyngeus in so far as I know is not understood, yet it is considered the commonest and most important etiological factor in the production of pulsion diverticula in that it produces resistance to the passage of the bolus from the pharynx into the esophagus without true organic stricture formation.

True stricture formation due to fibrosis at the cricopharyngeus level does occur and is an occasional etiological factor in the production of pulsion pharyngeal diverticula; this is illustrated by Cases 3 and 4. Dilations are necessary to relieve the dysphagia caused by stricture.

The alterations that take place in the hypopharynx due to the increase in size of the diverticulum are illustrated. The mechanical effect on swallowing is discussed.

The basic etiologic factors and the principles of repair are discussed, emphasizing the repair of the pharyngeal musculature with tailoring of the dilated hypopharynx to a funnel shape, in large diverticula, with the funnel opening through the relaxed cricopharyngeus.

Illustrative cases are presented to emphasize mistakes made in the surgical repair of pulsion diverticula and the application of the principles above mentioned to avoid them.

It is recognized that the surgically opened pharynx may be repaired and heal without the rest and protection afforded by tube and intravenous feeding. I recommend such precaution.

The technique and magnitude of the operative procedure for small and large diverticula must be varied to meet the anatomical variation, and operative treatment is recommended for small diverticula.

LAHEY CLINIC.

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LXXXI

ANESTHESIA IN BRONCHO-ESOPHAGOLOGY

A PANEL DISCUSSION

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The subject of anesthesia is vitally important; also controversial among the members of this group.

When visiting clinics in various parts of the country it has been noted that views reflected teaching background. At the time when there was one or two important centers of graduate teaching in broncho-esophagology, the subject of anesthesia could be taught simply and didactically. Topical anesthesia was the only accepted method. Any sort of basic narcosis, even open ether, in children, was considered dangerous. This conviction has been greatly modified.

The so-called method of "mummification" (no anesthesia) of children not in respiratory distress, even though they may have a foreign body in one bronchus, appears neither humane nor necessary. In my own experience, since I have used open ether in these cases, the incidence of postbronchoscopic tracheotomy has nearly reached a vanishing point. Our endoscopic forbears who taught us so much, and to whom we are so humbly grateful, did not have the capable assistance of the modern anesthesiologist.

It is not the purpose of this panel to make the subject less controversial but, rather, to stress the importance of a clear understanding of the well-established and practical procedures of all methods.

Broncho-esophagologists are deeply grateful to our anesthesiologists. They have taught us new and safer methods of anesthesia. Their scientific achievements of the last two decades have been phenomenal.

Anesthesiologists are now the skilled partners of the surgeons and bear equal responsibilities. We should never, therefore, attempt to bully them into doing something they consider unwise or unsafe.

It must be stressed that anesthesiology in our field does not differ greatly from that in others; respiratory and circular dynamics

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are essentially the same. Apart from a few anatomical factors the problem is identical. The objective is the same: A quiet anesthesia at a required plane, flexible and easily controllable, free from danger of explosion, and a quiet return to the protective reflexes so desirable in broncho-esophagology.

Table 1, taken from Hale's[1] *Anesthesiology*, shows a majority of 18 to 9 conditions in which pento-curare (general) was superior to local (regional) anesthesia. I am in accord with his views.

The surgeon's temperament, however, may make any one or several of the various anesthetic techniques or agents completely unsatisfactory, no matter how well applied. My own temperament demands a complete narcosis, unless there are positive contraindications making it undesirable or unsafe.

The tests for any suitable anesthetic in laryngo-broncho-esophagology are: 1) As complete abolition of the sphincteric action of the aryepiglottic folds and, 2) as complete relaxation of all the laryngeal and heavy neck muscles as safety will permit, while remembering that the heart muscles and cerebral blood vessels are also relaxed. Much trauma has frequently been inflicted on patients due to insufficient or improper anesthesia. This is particularly true with a nervous, struggling, gagging and unco-operative patient. The endoscopist cannot work efficiently under these circumstances; hence little, if anything, is accomplished.

I am of the opinion that we have all been schooled too much in using only topical anesthesia. Anesthesia, for any surgical procedure, must meet the following requirements: 1) The patient must experience no pain; 2) the patient must be sufficiently relaxed to permit accurate pursuit of the objective, whether it be surgical, diagnostic, or therapeutic; 3) the anesthetic must be safe.

With the majority of patients, the first two cannot be accomplished with topical anesthesia alone. All patients, except those with respiratory obstruction, cardiac conditions or other contraindications, should have both a topical and some form of a general anesthetic. It is quite certain, however, that no one anesthetic agent or technique is applicable or desirable for all patients. It must be selective and individualized. When, after consultation with the anesthesiologist, a complete narcosis seems advisable my routine, with individual modifications, is as follows:

For Children. Open ether (even with a foreign body in one bronchus). Premedication as indicated and as ordered by the anesthesiologist.

TABLE I
CHOICE OF ANESTHESIA

CONDITION (S—Superior) (G—Good) (U—Undesirable)	GENERAL				REGIONAL			REMARKS
	Pento Curare N2O-O2	Cyclo	Open Drop Ether	Ether Vapor	Spinal	Rrg. Block	Loc. Bl.	
Allergies and Asthma	G	G	S	S	G	G	G	Test for sensit. by skin tests
Anemia	S	S	U	G	U	G	S	High O² during op. Trans. during op.
Adrenal pathology	G	S	S	S	U	G	G	Adrenalin may be needed after tumor removed.
Brain pathology								Local good if
Acute accidents	S	S	G	G	U	U	S	uncons.
Psychotics	S	S	G	G	U	U	U	Uncoop. for locals
Tumors	S	S	G	G	U	U	U	High O²
Cautery to be used	S	U	U	U	S	S	S	
Children								
Under 1 year	U	S	S	U	U	U	G	Uncoop. for spinals
1–6 years	G	S	S	U	U	U	U	Uncoop. for spinals
6–17 years	S	S	G	G	G	U	U	
Diabetes	S	S	U	U	G	G	G	Cover glucose with insulin 1–4
Geriatric patients	S	G	U	U	G	S	S	No effects on B.P. with Pento-Curare High O² at all time
Heart pathology								
Hypertension	S	G	G	S	U	G	G	No adrenalin in local
Hypotension	G	S	G	G	U	S	S	
Coronary dis. sev.	U	G	U	U	U	S	S	
Decompensation	U	G	U	U	G	S	S	
Kidney pathology	S	S	U	U	G	G	G	
Liver pathology	G	S	U	U	G	S	S	Check bl. and cl. time during op.
Lung pathology								
Bronchiectasis	S	S	U	U	U	U	U	Explosion hazard
Carcinoma	S	S	U	G	U	U	U	when using cautery
Emphysema	S	S	U	G	U	U	U	in chest—high O²
Tuberculosis	S	S	U	U	U	G	G	needed in chest
Obstetrical patients	U	G	U	U	S	S	G	Heavy nupercaine pref. for spinals
Spinal cord injury or pathology	S	S	U	S	U	U	U	Don't use spinals because medico-leg. aspects. O.D. ether poor—need endotr. tube for prone pos.
Spleen pathology	S	S	S	S	U	U	U	Ether contracts P-C and Cyclo no eff.
Thyroid pathology & hyperthyroidism	S	G	U	G	U	S	S	In way with O.D. Ether
Vocal cord pathology	S	G	G	G	U	G	G	

For Adults. Nembutal, 1½ grains, or other barbiturate is given by mouth 90 minutes before the operation, morphine, ⅛ grain, and atropine grains 1/100 and/or scopalamine grains 1/200 are given by hypodermic injection intramuscularly. These are administered at least 45 minutes before the operation.

Twenty minutes before the operation the throat is sprayed three times at five-minute intervals with 2 per cent pontocaine or 10 per cent cocaine. The tongue is held forward.

Just before the intravenous anesthesia is started 2 cc of 2 per cent pontocaine, or 2 cc of 5 per cent or 10 per cent cocaine are dropped into the larynx slowly, drop by drop.

Coughing tends to distribute the anesthetic agent throughout the bronchial tree unless heavy bronchial secretions cover the mucosa.

The patient is taken into the operating room on schedule. Curare is given slowly in the vein, 6 to 12 mg, for the average sized patient. When the patient is unable to raise his head but a short distance from the table, ample curare for perfect relaxation has been given. Definite voice changes will also be noted and respiratory depression will be minimal or absent. The dosage of curare in muscular individuals is calculated as follows: 3 mg (1 cc) for every 50 pounds of body weight; for emaciated patients this is reduced to 3 mg per 75 pounds of body weight.

Pentothal sodium 2.5 per cent is given intravenously very slowly. It is advisable to give 1 or 2 cc of the above solution as a test dose to determine the patient's response. Allow 60 seconds to elapse before additional pentothal sodium is given. If there is no respiratory depression 1 to 2 cc increments are given until lid reflex is abolished. This takes three to five minutes. Oxygen is given continuously through a tube in the side of the mouth.

The surgeon must await the signal from the anesthetist before any endoscopic instrument is introduced. If resistance is noted, or if there is a sphincteric spasm of the aryepiglottic folds or the cricopharyngeal muscle, more local anesthetic must be sprayed on the area from an atomizer, the 'scope must be removed and more intravenous anesthetic used. When ample curare and local anesthetic are used, the dose of pentothal sodium is reduced by about one-half.

With mutual understanding between the surgeon and the anesthetist, as outlined above, the problems of anesthesia are usually solved.

SUMMARY

As stated in the beginning, the training area has much to do with the expressed opinions of the various discussants. The collective views tabulated from questionnaires circulated among Philadelphia endoscopists would vary greatly from those which might be circulated in Rochester, Minnesota, New Orleans, many cities in the middle west and on the Pacific coast.

The object of a panel discussion of this sort is to rehearse the indications and contraindications for the various techniques in anesthesia and the pharmacologic agents best suited to produce the desired result.

Premedication depends on the vigor of the patient. Standard preoperative orders are undesirable and may even be dangerous. It is better if in doubt to under-medicate.

The relaxants, such as curare, in divided doses by intravenous infusion, followed by sodium pentothal, seem to be gaining in favor each year among broncho-esophagologists.

Antagonists to the relaxants recently developed have greatly increased the safety margin in their use. Edrophonium (tensilon) seems one of the best to antagonize the curare compounds and restores the patient's protective reflexes after some spontaneous respiration has appeared. It may fail to overcome apne. Practicality makes its use justifiable even though its pharmacologic action may not be entirely clear. Small repeated doses are desirable. Oxygen must always be available.

Curare has a low pH and sodium pentothal has a high pH and must not be mixed but administered separately after cleansing the rubber tube with normal saline. Acid pentothal may form if the solutions are mixed.

Individual opinions and the temperament of the surgeon must govern to a great extent the selection of the anesthetic. The design of this forum has been to bring together the various opinions of experienced men so that we may all have better and safer anesthesia.

No matter what the technique or the anesthetic agent that may be used there are always hazards.

I shall continue, however, to maintain a dubiosity and be unwilling to accept the statement that the expert endoscopist may extract a foreign body from the bronchus of a frightened, struggling child without some degree of brutality and trauma. It is my avowed conviction that respiratory distress is the only justification for the "mummification" (no anesthesia) method.

Constant vigilant supervision of the patient by a skillful anesthesiologist will remain urgently important.

1103 GRAND AVENUE.

REFERENCE

1. Hale, Donald E.: Anesthesiology, By Forty American Authors. F. A. Davis Company, Philadelphia, 1954, p. 231.

JOHN T. STAGE, M.D.

JACKSONVILLE, FLA.

The purpose of my being on this panel is to present the indications and contra-indications in the use of general anaesthesia in the field of endoscopy. Let me preface any further remarks by stating that generally speaking a topical anaesthesia presents far less hazards than general anaesthesia in this particular field. I believe as a team, the surgeon and anaesthesiologist realize that this particular branch of surgery possesses inherent disadvantages found in no other field. These disadvantages are due to the fact that the surgeon and anaesthesiologist are interested and occupied in the same field and the anaesthesiologist must of necessity establish an adequate airway in one manner or another, while the surgeon is carrying on endoscopic examination or surgical maneuvers.

CONTRA-INDICATIONS

There are but a few actual contra-indications and many relative contra-indications to general anaesthesia in the endoscopic field:

1. *Hypoxia.* A patient, who because of some pathological condition is riding the thin edge of hypoxia, may be described by the surgeon as being unco-operative when endoscopic examination or surgery is attempted under topical anaesthesia. Actually the patient's oxygen reserve is so low that instrumentation in the tracheal bronchial tree may accentuate his hypoxia to such a state that he becomes irrational, restless, and unco-operative. In my opinion, this is the most dangerous patient of all because we are called occasionally to give general anaesthesia to this patient so that the surgeon may have better co-operation. This may turn out to be a fatal error in that anaesthesia merely accentuates the hypoxic condition and death may occur on the table.

2. *Emphysema.* The emphysematous patient who is physically disabled because of his condition is a poor candidate for general anaesthesia. I am not speaking of minimal emphysema as diagnosed by x-ray, but of the barrel-chested patient who complains of shortness of breath climbing stairs.

3. *Age.* The extremely aged and the very young are poor candidates for a general anaesthesia.

4. *Cardiac Damage.* A patient having suffered a recent infarct or one under treatment and with a history of cardiac damage should be viewed with a great deal of caution.

5. *Pulmonary Edema.* The wet lung is a poorly aerated tissue and complicates general anaesthesia.

INDICATIONS

1. *The Child.* With the exception of the extremely young, as stated above, the child probably deserves a general anaesthesia for the sake of himself and the surgeon.

2. *Patient's Preference.* The patient who desires general anaesthesia and in whom there is no contra-indication will be a candidate for general anaesthesia.

3. *Surgeon's Desire.* Due consideration should be given to a surgeon who desires general anaesthesia for his work because of the basis of time, of the desire to instruct during his work, or who feels that the examination or surgery will be benefitted by the advantage of a general anaesthesia.

TYPES OF GENERAL ANAESTHESIA

The field of anaesthesia offers but a few new drugs or methods that have not been explored in the last five years. The combinations used are inhalation anaesthesia, intravenous anaesthesia, rectal anaesthesia plus topical anaesthesia and curare. The use of topical anaesthesia with a general anaesthesia may be divided into topical directly or drugs used transtracheally. In the field of inhalation anaesthesia trichlorethylene is now being used in moderate amounts in combination with nitrous oxide and oxygen. The thiobarbiturates such as Pentothal® (Thiopental Sodium) or Surital® (Thioamylal Sodium) still hold sway in the intravenous fields. The rectal route has not offered any new agents and rectal Pentothal (Thiopental Sodium) still holds the top rank in this field. Two topical anaesthesias, Xylocaine® (Lidocaine Hydrochloride) and Cyclaine Hydrochloride® (Hexylcaine Hydrochloride) have proven to be of considerable value. Antihistamines have been suggested as topical anaesthesia for endoscopic work, but in my opinion they have turned out to be irritating and of inadequate depth. The curares which are of course not anaesthetic agents, but are supplemental paralyzing agents, have had the addition of Succinylcholine® added to their field. This drug has proved to be of interest and of value. It is an agent which used as a continuous intravenous drip can produce any stage of relaxation in a matter of seconds and this effect can be erased by discontinuing the drug. At present, we have no antagonist for this curare-like agent. It does offer the possibility of quick recovery following surgical procedure.

It is impossible to present in detail any segment of anaesthetic techniques on such a panel unless requested. Every anaesthesiologist has his pet drugs and procedures and there are many correct ways to do one procedure. The problems which may arise in their field are common to all. Perhaps these problems have worried you as they have troubled me and I have listed them in the hope that a discussion of them will give all of us help. Here are a few:

1. The asthmatic patient.
2. Laryngeal edema and the endotracheal tube.
3. Granuloma and the endotracheal tube.
4. Preoperative cortisone and ACTH.
5. Postoperative use of cortisone.
6. The dangers of Thorazine.
7. Cardiac arrest and fibrillation.
8. Nasal, oral and tracheal reflexes.
9. Convulsions—topical and general agents.
10. The curares and prolonged apnea.
11. Laryngeal and bronchial spasm.
12. The bleeding problem patient.

In conclusion and most important of all, it is my belief that a patient who undergoes a general anaesthesia for this type of work should have as careful a preoperative and postoperative evaluation and medication as those undergoing any other type of surgery or examination. At no time, should the surgery or the anaesthesia be examination. At no time, should the surgery or the anaesthesia be considered to be of a minor nature. Secondly, I believe that this patient tion during the recovery period and in this respect I believe our modern recovery room will prove to be of extreme value.

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PAUL H. HOLINGER, M.D.

CHICAGO, ILL.

There is almost as great a variety of opinion in regard to anesthesia in peroral endoscopy as there are peroral endoscopists. One reason is the difference in evaluation of the effectiveness of techniques by different individuals. Another is the degree of anesthesia demanded by different surgeons; what may be satisfactory anesthesia for one is entirely unsatisfactory for another. The effectiveness of the operating team also influences the choice of anesthetic either the surgeon or the anesthesiologist wishes to use. And finally, such basic problems as the age of the patient, the duration of procedure, the associated pulmonary or cardiac pathology, the lesion for which the examination is being made, the actual or potential respiratory obstruction and the patient's preference all play a part in the determination of the suitability, effectiveness and ultimate choice.

Our own routine for anesthesia in peroral endoscopy follows the principles and techniques suggested by the Jacksons. Local anesthesia is used almost invariably in adults. It consists of spraying the pharynx with 2 per cent pontocaine and then instilling 2 cc of 10 per cent cocaine into the larynx and trachea with a laryngeal syringe under mirror laryngoscopic guidance. The spray is directed first on the tongue, palate and posterior pharyngeal wall, then with the patient in the sitting position it is directed into the larynx as the tongue is held forward and the atomizer tip directed downward. Three to five minutes is allowed to elapse between the spraying and the laryngeal instillation; toxicity is based not alone on total dosage, but also on rate of absorption. It is well known that a specified amount of anesthetic given in a very short time may cause a reaction, whereas if administered in divided amounts over a considerably longer period of time it is not only more effective but is less likely to cause a reaction. In our experience, both with our own trials and the observations in other clinics, aerosol administration has been unsatisfactory.

Preoperative medication depends on the age and general condition of the patient. None is used in old or debilitated individuals, or those with respiratory obstruction. In routine cases, adults, 50 mg of Demerol® and 2 grains of sodium phenobarbital are given hypodermically approximately one hour preoperatively. Atropine is used in addition if the patient is to have a laryngoscopy or esophagoscopy, but generally omitted for bronchoscopy since the procedure

so often is performed to obtain secretions for bacteriologic or histologic examination; this aspect of the examination would be defeated if atropine were administered.

The use of general anesthesia in peroral endoscopy is complicated by the fact that both endoscopist and anesthesiologist must compete for the airway. Each is responsible for the maintenance of a patent air passage, but with a laryngoscope or bronchoscope in place for a surgical endoscopic procedure, the anesthesiologist can no longer continue respiratory movements for the patient should these have been abolished through the action of curare or a similar agent. In direct laryngoscopy performed for the removal of benign lesions such as polyps or nodules, a more satisfactory evaluation of the end result can be obtained if the patient is conscious and can phonate on request during the procedure. If the larynx is unusually difficult to expose, a minimal amount of curare may be administered during the procedure to permit adequate relaxation for satisfactory larynx exposure, while still maintaining the ability to phonate on request of the operating surgeon.

Local anesthesia for esophagoscopy is identical to that used for direct laryngoscopy. If a general anesthetic is to be used the patency of the airway must be maintained with an intratracheal tube. This holds true for adults and children since the esophagoscope can compress the trachea sufficiently to cause asphyxia.

As a rule, we use no local or general anesthesia for endoscopic procedures in infants or children. Adequate sedation with morphine or Demerol and a barbiturate is used in children unless respiratory obstruction contraindicates its use. Exceptions to this rule are procedures anticipated to be prolonged; open drop ether is most satisfactory for laryngoscopy under these conditions. For esophagoscopy an intratracheal airway is absolutely essential and the choice of the agent may rest entirely with the anesthesiologist. Open drop ether is also most satisfactory for bronchoscopy in children who are to be examined under general anesthesia, and some advocate it for bronchography as well. We prefer to do our bronchography under local anesthesia in adults and in children we use no anesthesia, but employ adequate sedation. No anesthesia or sedation is used for this procedure in infants, and a laryngoscope and an aspirator are available in the fluoroscopic room to aspirate the oil if its viscosity causes obstruction in the tiny infant's airway.

Returning to the problem of evaluating the effectiveness of a local anesthetic, we find so many variables that for accurate, thorough study of a new agent, all possible variables and responses should be

TABLE I. ENDOSCOPY TOPICAL ANESTHETIC STUDY SHEET

NAME REG# DATE AGE SEX WT. HT. RACE WARD PHYSICIANS SIGNATURE

PHYSICAL DISEASES — COMMENTS REGARDING SUITABILITY OF PATIENT FOR ANESTHESIA — PRE-ANESTHETIC IMPRESSION — POST-ANESTHETIC IMPRESSION

ALLERGIES

PRE-ANESTHETIC MEDICATION — DRUG / DOSE / ROUTE / TIME — TIME ANESTHETIC ADMINISTERED — TIME PROCEDURE STARTED — DURATION OF PROCEDURE ___ MINUTES — PROCEDURE: BRONCHOSCOPY, LARYNGOSCOPY, ESOPHOGEAL DIL., OTHER

TOPICAL AGENT* — AMOUNT CC. — CONC.% — VEHICLE: JELLY, SOLUTION — ANY SUPPLEMENTARY ANESTHESIA: GENERAL, OTHER — REASON:

QUALITY ANESTHESIA: EXCELLENT, GOOD, FAIR, POOR, NONE — PAIN: SLIGHT, MODERATE, SEVERE, NONE — SENSATION: SLIGHT, MODERATE, MARKED, NONE — ONSET: RAPID, SLOW, WITHIN ___ MIN. — DURATION ADEQUATE: YES, NO, AT LEAST ___ MIN.

REFLEXES	MARKEDLY OBTUNDED	SLIGHTLY ACTIVE	MODERATELY ACTIVE	NO OBTUNDATION
LARYNGEAL				
CORINAL				
COUGH				

BUCKING: NONE, SLIGHT, MODERATE, SEVERE

EFFECTS AT SITE OF APPLICATION: BURNING NO__ SL.__ MOD.__ SEVERE__ OTHER: DESCRIBE

TASTE

PRE-ANESTHETIC: PULSE /MIN. B.P. / mm Hg RESP. /MIN.

POST-ANESTHETIC: /MIN. / mm Hg /MIN.

IRREGULARITIES DESCRIBE:

REACTION: YES, NO, IMMEDIATE, DELAYED

TYPE REACTION: CHECK THOSE THAT APPLY

NAUSEA, BRADYCARDIA, TACHYCARDIA, APPREHENSION, NERVOUSNESS, TALKATIVE, TREMORS, CONVULSIONS, UNCONSCIOUSNESS, SHOCK, CARDIAC ARREST, DEATH, PSYCHIC, OTHERS: DESCRIBE

REVIEW AS TO CAUSE AND TREATMENT (SEVERE RZACTIONS)

* DRUG CODE KEY_____

M-593

recorded. The evaluation chart (Table I) was prepared in conjunction with Dr. Max Sadove, head of the department of anesthesiology of the University of Illinois. It may seem unduly complicated, but a careful study of any new local anesthetic agent for peroral endoscopy should include consideration of all the listed questions.

700 N. Michigan Blvd.

CHEVALIER L. JACKSON, M.D.

PHILADELPHIA, PA.

I felt a considerable responsibility in receiving this assignment, and in view of the fact that we have five teaching clinics in Philadelphia which are on good terms with each other and well acquainted, yet by no means using exactly the same techniques and the same methods, I though it would be well to resort to the old questionnaire method of preparing a paper, which is very nice, because it enables you to get far along the way with the help of others. I felt that in this particular instance it would be really valuable to present not just my personal views, but the consensus of views and a summary of current practice in Philadelphia clinics. Most of those who answered my questionnaire are here in the audience, and I hope they will participate directly in the discussion later. Hahnemann, Jefferson, the University of Pennsylvania, Woman's Medical College and Temple are all represented.

In the paper, I am going to tabulate all the information received, including the specific doses and percentages used by the different bronchoesophagologists who answered the questionnaire.

The following five fundamental broad questions were asked:

1) Do you prefer local or general anesthesia for endoscopic procedures?
2) State briefly the indications for use of general anesthesia as you see them.
3) What drugs and dosage do you use for: a) premedication; b) local anesthesia?
4) If you use general anesthesia, what form do you prefer?
5) If you have had any complications in connection with the use of either general or local anesthesia, please state them.

The answers to the first question were simple, because everyone, in one word, answered "local" in reply to the question. As for the replies to the second question, there was a substantial agreement on the indications for general anesthesia, and I might list here the indications gathered from the various replies. In the first place, anatomical difficulties presented by the particular patient; secondly, large foreign bodies in the esophagus; thirdly, unco-operative or mentally

unfit patients; fourth, suspension laryngoscopy; and finally, the patient's insistence. "Direct laryngoscopy in the heavy-set, thick-necked individual" was mentioned by several, and one of the answers was, "Whenever tonsillectomy and adenoidectmy, or some other procedures were to be done at the same time."

The answers to the third question, as to what drugs and dosage was preferred, have been summarized in a table. Nembutal® and Demerol® were mentioned in almost all of the answers. The opiates, both morphine and pantopon, were mentioned, and atropine was used by one or two. As far as sprays and instillations were concerned, I think it is important to state that most seem still to use some cocaine, in 1 per cent, 4 per cent or 10 per cent solution. A word of warning was generally added in connection with the use of 10 per cent solution, that it should be "fractionally" used in a limited quantity. The most interesting remark about percentages was in connection with pontocaine. We have been accustomed to use 2 per cent pontocaine rather liberally, and I was surprised to find that Dr. Clerf stated very clearly that he used 0.5 per cent pontocaine. He stated that since using 0.5 per cent solution he had not seen any toxic effects, but that he had seen toxic effects using a 2 per cent solution. Dr. Putney is of the same opinion. Xylocaine® is the one new drug that was mentioned, and we at Temple are among those who have been using it with considerable satisfaction, especially in children.

The fourth question was, "If you use general anesthesia, what form do you prefer?" Two out of eight preferred pentothal, one pentothal with curare, four ether; and one specified ether in children and pentothal in adults.

The fifth and last question on the questionnaire was with regard to complications, and it was gratifying to hear how few complications were reported. One death was supposedly due to pontocaine used for a bronchogram, and one death possibly due to cocaine, but in a case in which there was considerable doubt as to whether the cocaine was responsible. The other complications were mild reactions only, to cocaine and to pontocaine.

To state very briefly our own feeling, Dr. Norris and I at Temple, of course, prefer local anesthesia for most cases. Our indications for the use of general are much the same as those of others. I would recognize all the indications that I have mentioned as summarized from the different answers. As far as premedication is concerned, we have been using Nembutal, Demerol, and some atropine. For direct laryngoscopy, we use a 2 per cent pontocaine spray, and then several fractionated instillations of 10 per cent cocaine instilled under

the guidance of the laryngeal mirror. When we have used general anesthesia, we have used anectine and pentothal, and we have also used ether. I think that the removal of nodules, or small tumors of the larynx in young children, is certainly one of the important indications that I have recognized myself in a number of cases. To remove a vocal nodule from the cord of a young child, I think that ether anesthesia is very satisfactory.

I would like to say, before concluding, that we do believe that technique of administration of local anesthesia is important. We use first a spray, then a fractionated instillation under the guidance of the mirror, instilling a few drops of the solution into the larynx; waiting, and then instilling a few more drops, and then a few more drops—about three fractionations. We feel that proper training is very necessary to obtain a satisfactory effect. The old technique of applying anesthetic solution on a sponge to the pyriform sinuses by means of a curved "cross-action" forceps, so that it would anesthetize the superior laryngeal nerves, was discarded many years ago as being inconvenient and unpleasant, and no more satisfactory than fractional instillation.

We still believe that the great majority of work in the endoscopic clinic is better done under local anesthesia, or in the case of infants and young children, without anesthetic. We believe that there is no doubt but that in the early days of the development of endoscopy, one of the great advances in safety and efficiency was the development of a technique which would permit doing these procedures without anesthesia. Of course, we agree that the risks have been so greatly reduced by the developments of modern anesthesiology, that the indications should perhaps be considerably broadened at the present time. However, we still insist that if we have to work in the dark, as for example under fluoroscopic guidance, local anesthesia or no anesthesia is much safer. We feel that it is safer also, when there is possible respiratory obstruction, either from narrowing of the airway by a lesion or by a large foreign body or lesion in the esophagus. And then also, we believe it is much simpler for the endoscopist, for the nurses, and certainly for the patient as well, if the procedure can be done smoothly and satisfactorily under local anesthesia. Furthermore, in closing I should like to say that we must not lose sight of the great importance of *vocal* anesthesia—by which I mean the psychological preparation of the patient who is to be examined or endoscoped under a local. Then, in the case of a child, I should like to stress the importance of proper control of the patient. This does not mean just holding him down and going ahead with the endoscopy; but it means that if you have the child

laid on a cover, and the cover wrapped over the arms and under the back and the head is well held, that you get on with the procedure and get through with it almost before the patient or the endoscopist knows that it is being done, whereas if you try to hold a child too young to co-operate, and hold him inadequately, the procedure will indeed be dangerous, and painful besides! I think that the control of the child belongs right in with a discussion of anesthesia. That may seem paradoxical, but I believe it is true!

3401 N. Broad St.

DISCUSSION FROM THE FLOOR

DR. CHARLES S. BLASE (Cincinnati, O.): I would like to have one or several members of the panel comment on transtracheal block.

During a clinical investigation of cyclane anesthesia, we have thus far performed about 400 bronchoscopies using the transtracheal block. These cases have, for the most part, been done in an institution for tuberculosis where adequate follow-up care could be given. Five per cent cyclane was used exclusively in this series. Thus far, we have obtained no untoward reactions and have also felt that there were definite advantages in the transtracheal route for the application of local anesthesia for bronchoscopy.

Would the panel please give their opinion on the transtracheal route.

DR. JOSEPH P. ATKINS (Philadelphia, Pa.): I would like to second Dr. Jackson's point that the control of the child is an important part of the anesthesia. The problems that we had in the Children's Hospital in Philadelphia disappeared completely when we insisted on having a team that could satisfactorily control children. We have no need for elaborate premedication or anesthetic care.

On a few occasions we have encountered foreign body problems in which it was necessary for the child to be particularly quiet. These children were premedicated, and endoscopy was then carried out with the aid of one of the curare-like drugs to secure better relaxation. Most of these were esophagoscopies. The curare-like drugs were administered by the anesthetist who controlled respiration by means of an endotracheal tube through which oxygen was insufflated. These children got along very satisfactorily from the emotional standpoint and the use of strong central nervous system depressants was avoided.

From time to time we use cyclane and get satisfactory anesthesia. It is slower than cocaine but we like to use it on the patients who claim that they have had a bad previous experience with cocaine.

It is my very strong feeling that most of the toxic manifestations of local anesthetics are due to over-dosage rather than sensitivity. I would like to know what the feeling of the panel is with regard to this.

DR. JOHN J. O'KEEFE (Philadelphia, Pa.): I hope I am not talking out of turn when I bring this thought to the attention of the audience in considering the subject of anesthesia in endoscopy to consider it from two aspects: First is a consideration of the amount of time and effort necessarily expended in obtaining anesthesia in the difficult patient: The child, the aged, or the debilitated. Such anesthesia can be obtained in these patients if we make the effort, using any one of the topical anesthetic agents. Most of us in larger clinics favor the use of topical anesthesia, because in our hands it has been effective. It is quick in its action. It is relatively nontoxic and recovery is quick. A large number of patients can be examined with the least difficulty and inconvenience both to them and to us.

Another though is that those of us who are teaching must rotate our patients relatively quickly through the clinic so that a large number can be demonstrated.

The disproportionate amount of time necessary for general anesthesia is not compatible with this type of teaching.

DR. WALTER H. MALONEY (Philadelphia, Pa.): I would like to add a few remarks in addition to the data submitted on Dr. Jackson's questionnaire.

When to change from local anesthesia to general anesthesia is the subject most thoroughly discussed in the postgraduate course in broncho-esophagology. I feel

that it is a responsibility of the bronchoscopist to develop his technique with local anesthesia to the highest degree so that when the situation arises in which general anesthesia is contraindicated he will be able to afford the patient the best in endoscopic technique. Therefore, I would urge anyone doing endoscopy to elect local anesthesia and use general anesthesia only in those cases in which there is a definite indication.

I think the biggest factor in an unsuccessful local anesthesia is the lack of attention to detail. It is the indiscriminate change in established routine which results in the bronchoscopy being traumatic and unsatisfactory. We are tempted to substitute newer drugs not thoroughly evaluated, and to condemn local anesthesia instead of the new drug when the anesthesia is unsatisfactory.

Regarding the preoperative use of atropine I feel that the local anesthesia is more effective on a slightly dried mucous membrane than on one protected by a thick coat of secretion. One must also consider the effect of atropine on the vagus nerve, and in patients with cardiac disease I feel that the action of atropine is particularly needed.

Dr. F. Johnson Putney (Philadelphia, Pa.): I might say just a few words about pontocaine, since we are the only clinic in Philadelphia that uses it exclusively, and Dr. Jackson referred to that fact. I would like to make our position very clear about the use of the percentages he mentioned.

The use of 2 per cent pontocaine has been given up because I feel that pontocaine is a very toxic drug when used in that strength and in the amounts necessary to obtain anesthesia of the tracheobronchial tree. In fact, I hesitate to use 2 per cent pontocaine at any time and in any amount. One-half per cent solution has provided satisfactory and adequate anesthesia and at the same time has proved much safer. The amount of 0.5 per cent pontocaine used on any patient does not exceed 8 cc.

As an example of the importance of the percentage, I might cite a patient who had been coming into our clinic for years for the treatment of bronchiectasis. We do not have many of these patients any more, but a few are still around and this woman had been receiving periodic bronchoscopy for approximately four years and had never had a reaction from the anesthetic which was 0.5 per cent pontocaine solution. About two weeks ago, after administration of the anesthetic in the same dosage, she had a severe reaction prior to bronchoscopy. The question came up, had she developed sensitivity to pontocaine? On checking back we found that the resident had substituted 2 per cent pontocaine for 0.5 per cent and she was the first patient that had received any of the 2 per cent solution, with the resulting immediate reaction.

Dr. Edwin N. Broyles (Baltimore, Maryland): I would like to ask the panel if we have had any experience with aerosol methods of the instillation of local anesthetics through the very fine spray at the beginning of the local anesthesia. I might also say, Dr. Putney, that I remember one patient who developed severe asthmatic attacks with the use of pontocaine, and as a matter of fact we couldn't go on with the bronchoscopic examination; whereas a couple of days later by using straight cocaine, 10 per cent and even 20 per cent, he had no asthma at all, and we could finish the examination.

Dr. W. Likely Simpson (Memphis, Tennessee): I have heard the rumor that curare was dangerous to give in anesthesia. I think this panel ought to answer that.

Dr. Herbert W. Schmidt (Rochester, Minnesota): I would like to ask this panel just what type of anesthesia they use in children when they are doing bronchography?

Dr. Murdock Equen (Atlanta, Georgia): I would like to ask Dr. Stage to discuss cardiac arrest. Apparently, these cases of cardiac arrest have received a mul-

tiplicity of drugs, and I heard an internist say recently the more drugs a patient receives, the greater his chances are for having cardiac arrest.

I would also like to ask the members of the panel if they have ever seen a case of cardiac arrest when only ether had been administered.

DR. JOHN STAGE (in reply): I will limit my discussion to those questions pertaining to general anesthesia. The first question was in regard to the etiology of cardiac arrest. Study of the literature points to the fact that there are primarily three schools of thought: One school points to the anoxia, one school to multiplicity of drugs, and the third believes reflexes may be the source of cardiac arrest. All of us in the field of anesthesia doubt if one theory alone is correct. If I were forced to select the most prominent of the three, I would consider anoxia as the most outstanding.

The statement "multiplicity of drugs" has become such a well-worn cliché that any anesthesiologist using more than one agent feels as if he has committed a mortal sin. Personally, I doubt that our surgeons would really desire to return to the days of a choice between local anesthesia, spinal anesthesia, or straight ether. I would like to refer to a report I made of 11 cases of cardiac arrest in this area. In the majority of these cases, a single anesthetic agent was used, and ether led the list. If one looks back, searching honestly, for the most common etiological factor apparent in the arrests that have occurred under one's own care, there is no denying the fact that, in the majority of cases, there has been a period of hypoxia. One must not believe that it is an easy thing to ascertain hypoxia in a patient undergoing anesthesia in surgery. I will continue to use what is referred to as a "multiplicity of drugs" because I firmly believe that good anesthesia is balanced anesthesia. I believe just as firmly that the use of many drugs to counter-balance poor anesthetic technique can be a cardinal sin in our field.

Question number two was regarding the dangers of curare. Curare is not an anesthetic agent, but produces relaxation only. This relaxant comes in two forms. One we can counteract and one we cannot. The one we cannot counteract can only be combated by inflation of the lungs until the drug is destroyed. Occasionally one meets a patient in whom either type of relaxant will have a prolonged effect. The newest curare-like drug, succinyl choline, if used by a continuous drip method, appears to be one of the safest at present. The drug is destroyed so rapidly in the body that one can practically "titer" the degree of paralysis by the rapidity of the intravenous flow. If I were to establish a simple rule for the use of curare, I would state that only under unusual circumstances is apnea produced by curare. The anesthetized patient presents two important vital signs—pulse and respiratory movement. When pharmacological tampering destroys one of these signs, then we have lost fifty per cent of our chances of keeping the patient in physiological balance.

Question number three was in relation to the transtracheal route for topical anesthesia. The transtracheal route for topical anesthesia has one objection which has not been presented this afternoon. I have seen two deaths under topical anesthesia for bronchoscopic examination. Neither patient received the agent by the transtracheal route, but I doubt if the results would have been any different. The point I am presenting is this: The patient whose reflexes are obtunded under heavy medication may present a suppressed cough reflex. The transtracheal route having by-passed the larynx makes it even more easy to place topical anesthetic agents deep within the bronchial tree. In the two patients mentioned, catheters had been placed between the vocal cords and the patient was placed in a sitting position. The agent, pontocaine, then was injected rapidly through the catheter into the tracheobronchial tree, and, because of the heavy medication suppressed the patient's cough, the drug passed into the smaller bronchioles and across the two membranes separating the drug from the blood stream. It might be well not to place the patient in the sitting position if the patient has been heavily medicated for a transtracheal

approach as it can be seen that a perpendicular respiratory tree will drain topical anesthetic drugs into the alveoli more quickly than if the patient were placed at a less acute angle.

The question has arisen as to the possibility of dosage being the cause of reactions to topical and local anesthetics. The patient presenting a reaction to a topical or a local anesthetic agent will react in one of two ways, either he will convulse or he will show the typical picture of shock with hypotension, a pallid skin and a rapid or absent pulse. He may show a combination of both. Both patients need oxygen. The patient who reacts with convulsion needs intravenous barbiturates and the one with hypotension needs a vasoconstrictor. We are warned that there are patients who are so sensitive to a local anesthesia and the reaction is so immediate that death may ensue before any type of therapy has been started. In my opinion, these are rare compared to the two above types of reaction.

The closest one can approach an experimental condition with a local anesthetic is the use of intravenous procaine for various conditions in patients. As one increases the intravenous flow of procaine in the patient, he will state that he feels a "going away" sensation, a brassy taste and a feeling of warmth spreading over his body. He may even develop mild hypotension. If at this point the procaine solution is not slowed, the patient may develop increasing hypotension or show convulsions. I believe that this is as precise a test as one will see for the proof of dosage in local anesthetic agents on a patient. A second factor, speed, is harder to evaluate. Speed of absorption is based on several factors; the vascularity of the area injected, the dosage injected and the use of vasoconstrictors in the local anesthetic agent. One must realize that there is no way of checking as to whether a patient will tolerate the usual dosage and one has no idea of how quickly he will destroy the dosage offered him. With these unknowns, regardless of one's theories as to reactions to local anesthetics, all the anesthesiologist or surgeon can do then is to offer the usual protection; i.e., barbiturates prior to the use of local anesthetics, the use of vasoconstrictors in these agents when the vasoconstrictor is not contraindicated, the careful control of the dosage according to the patient's weight and age and being prepared with oxygen, vasoconstrictors and intravenous barbiturates to counteract the effect of a reaction when it does occur.

DR. PAUL H. HOLINGER (in reply): First I want to compliment Dr. Stage for a statement he made which is so important to all of us. He said he has discontinued "taking a patient down" with curare where breathing is out of the patient's control; he no longer feels that he has "done something" when he can tamper with the patient's physiology sufficiently to take away his breathing action and then give it back to him at will. Under some circumstances this is necessary, but those of us who have gone through this have seen a young anesthesiologist do this without real reason, and I think we all know what the problem is. As somebody said, it is as though he had a brand new, high-powered car, and was going to see how fast it could go, and sometimes the brakes were put on just a little too late. Fortunately, there has been a change in this attitude.

In cases in which we wish to use curare, we prefer to use a minimum amount, and only after adequate topical anesthetic has been applied. This is particularly effective for direct laryngoscopy in those patients who have a difficult anatomical configuration of the mouth. Under these circumstances a little curare permits exposure of the larynx in a very satisfactory manner. To put that patient asleep with pentothal, obtain relaxation with curare, and then try to work on the larynx has often been a nightmare, because these patients are heavy-set patients with the short necks, in whom most trouble will occur. Then a terrible emergency arises in which an exposure of the larynx has to be made at all costs and an intratracheal tube forced through the spastic cords with the patient deeply cyanotic from the obstruction. The problem of trying to do any laryngeal surgery such as the removal of a polyp is obviously absolutely impossible.

Transtracheal anesthesia has been used and is advocated more and more by anesthesiologists and by some roentgenologists for bronchograms; I believe that most of them are using it successfully. There are several objections. One, the possibility of infection through the cricothyroid membrane. A second is the possibility of injection of the material into the soft tissues of the neck. The discusser, Dr. Blase, just reported 2000 cases, I believe, without complications. In his hands, obviously, this is a very good technique. In other hands, if one studies bronchograms made through transtracheal injection, one will see lipiodol in the mediastinal structures. There is also the real possibility that the local topical anesthetic may be injected into the soft tissues of the neck as the patient coughs.

The question of reaction to local anesthetics is not always on the basis of dosage. I would disagree with Dr. Putney on that problem. There was a very excellent study made in one of the large veterans' hospitals in which it was found that all the reactions, or practically all of them, occurred in one branch of the service; none had occurred in the otolaryngological branch of the service. This showed that it is a problem of learning the technique of administration, using it properly, and observing the patient rather than continuing on a predetermined dosage as so often occurred in other services doing endoscopic procedures.

Aerosol anesthesia was mentioned by Dr. Broyles. We tried it ourselves, and we find it highly unsatisfactory, although it has been described as a very satisfactory procedure. The patient is simply given an aerolator with a certain amount of pontocaine in it; oxygen is attached and the patient is instructed to sit and inhale this; the procedure is performed after he has finished inhaling it. The surgeon himself could be busy about something else, another case, or whatever may be. In one clinic in which we saw this technique being used, the surgeon was highly pleased with the effectiveness of this form of anesthesia. From our standpoint, we never would haev dared to continue to do a laryngoscopy on a patient who had had that ineffectual an anesthetic. The procedure that was performed could not have been considered satisfactory in any way.

One sees again how variable the evaluation of the degree of anesthesia can be. I think the important point is to discontinue the procedure if the anesthetic is unsatisfactory and reanesthetize the patient. Do not be afraid to stop and add a little more anesthetic, provided the patient shows no sign of reaction, such as talkativeness, restlessness, and so forth.

We have argued about anesthesia for children for bronchography so often before this Society. If Dr. Wishart were here, we could start the whole argument over again. He uses ether. We do not use ether. It is just as simple as that. In older children we prefer to give morphine, and do the bronchography without anesthesia. I believe Dr. Clerf also mentioned a year or two ago he preferred ether anesthesia for bronchography in children.

In small infants, no anesthesia is necessary. In any child, I think it is important that a laryngoscope, an aspirator and a suction machine be present in the fluoroscopic room when the oil is being injected, because sometimes respiratory obstruction occurs due to the viscosity of the oil and the small size of the infant's trachea and bronchi. A suction pump is a life saver under these circumstances.

Dr. Chevalier L. Jackson (in reply): First, with regard to transtracheal block: I have been one of those who has always advocated the broadening of this Society and its membership to include broncho-esophagologists who come in from all angles, anesthesiologists, gastroenterologists, thoracic surgeons, internists, and so forth. But I think that every member should be to some extent a laryngologist. I think that the local anesthesia of the larynx and tracheobronchial tree should be performed through the mouth. I agree entirely with Dr. Holinger about the possible complications, which may be avoided by perfect technique, it is true, but which are nevertheless objections. The examination of the pharynx and larynx is a very

important preliminary to an endoscopic procedure, and the anesthetization of the pharynx and upper larynx is just as important as the anesthetization of the tracheobronchial tree. So, as Dr. Holinger says, people are getting away with it, but I don't think we can go on record as approving it.

I was really hoping that Dr. Stage would say something about the relation of dosage to toxicity. Maybe he may still say a word on that question, because I think he is best qualified to speak. Personally, I might always have been under the impression that even a very small amount of the drug was sufficient, if the patient had an idiosyncrasy for it, to cause a severe or even fatal reaction. I would like to know what Dr. Stage thinks about that.

There are just two other points that I want to mention. I might say that Dr. Maloney has clearly stated that he does not feel obliged to agree with me, and I might say that that feeling is mutual! As far as his being the only one to mention the use of atropine, however, in reply to the questionnaire, I stated that only "some" used atropine. They all used these other drugs. The Temple Clinic is among those who do use atropine. We have consistently used atropine from the beginning. While Dr. Maloney naturally has the cardiac reasons predominant in his mind, in my mind the lessening of secretions in the pharynx has been the chief consideration, particularly in anesthesia for direct laryngoscopy. The diminution of oral secretions that you get from atropine makes it much easier to do a direct laryngoscopy.

Finally, Dr. Broyles mentioned the toxicity of pontocaine in cases of asthma. I might say that in Dr. Clerf's and Dr. Putney's replies to the questionnaire, while they were very much in favor of pontocaine in the small dosages, they did say "except in allergic and asthmatic cases." They do not favor its use in those cases and neither do we.

LXXXII

SELF-RETAINING ESOPHAGEAL INTUBATION TUBE

PAUL H. HOLINGER, M.D.

CHICAGO, ILL.

Numerous metal and plastic tubes have been suggested to assist in maintaining the esophageal lumen in cases of inoperable carcinoma of the esophagus. This tube is a modification of one presented two years ago at the meeting of the International Bronchoesophagological Society. It is obtainable in sizes 22, 26 and 30 F., and lengths of 3 to 5 inches. The tube is made of polyvinyl, a radiopaque plastic tubing. Both ends are open. Close to each end a mushroom catheter type flare holds the tube in place.

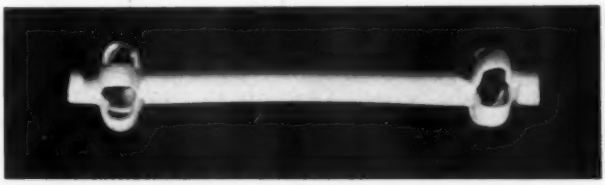


Fig. 1.—Polyvinyl tube used to maintain a lumen through an inoperable esophageal carcinoma.

These tubes are inserted by passing a 9 mm full lumen esophagoscope through an obstructing carcinoma using a string guide, if possible. The tube is then passed through the esophagoscope until its distal end protrudes from the scope and becomes anchored at the lower end of the tumor. As the scope is withdrawn, the tube is pushed out of the scope with forceps leaving the proximal mushroom at the upper end of the tumor. Thus, this type of tube cannot be regurgitated and cannot inadvertently pass into the stomach. Because the tubes are radiopaque their position can be checked, and in special problems of introduction the procedure can be fluoroscopically guided.

These tubes are manufactured by the American Cystoscope Makers, Inc., of New York.

700 N. MICHIGAN AVE.

LXXXIII

DISTALLY AND PROXIMALLY LIGHTED ENDOSCOPES

Dr. Edwin N. Broyles, M.D.

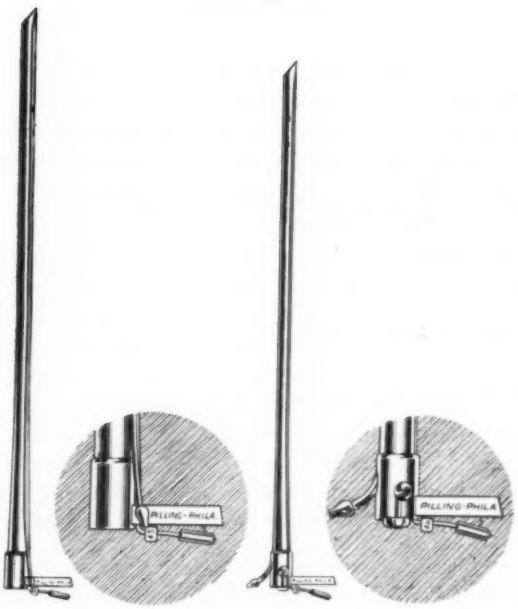
Baltimore, Md.

I would like to demonstrate a set of endoscopes which have combined distal and proximal lighting. These 'scopes are shaped somewhat like Mr. Negus' 'scopes. They have a blower on the bronchoscope that prevents fogging of the operator's glasses or shield; also a small oxygen tube that supplies oxygen in cases of difficulty with breathing. For examination, the brightest light that I know is the Bruning's handle light. We have adopted this Bruning's handle to fit on the proximal end of the 'scopes.

The distal lighting can be used without the proximal lighting and, if more illumination is needed than the distal light will give, the Bruning's handle can be attached easily and quickly. When lighted it gives a powerful concentrated light down through the tube, so that the operator can see beyond, not just in the close proximity of the distal end of the 'scope. Certainly in some types of cases for observation the proximal lighting is a great aid. For operative procedures the distal lighting is the only thing to use and so, with these types of instruments with both proximal and distal lighting, we can see and learn things that we have not been able to in the past.

These 'scopes are made in all sizes, beginning with a 3½ millimeter bronchoscope; also there are different sizes of esophagoscopes. They are manufactured by Pilling and are to be called "The Universal Endoscopes."

1100 N. Charles St.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Clinical Notes

LXXXIV

REPAIR OF A PERSISTENTLY PATENT GLOTTIS:

REPORT OF A CASE

WILLARD PARKER, M.D.

CLEVELAND, O.

In the recent literature there has been much emphasis on the surgical treatment of abductor paralysis of both vocal cords with a persistently closed glottis; but very little has been written about the opposite condition, in which the glottis is persistently open. This latter condition admittedly is uncommon, but when it does occur it may present a difficult problem.

The case report that is being presented here, is an example of a persistently open glottis with loss and distortion of the supraglottic structures on one side. It is of special interest because of the medical and surgical problems involved, and also because it demonstrates the important role of the supraglottic structures in protecting the lower airway from the aspiration of food and fluids.

REPORT OF A CASE

A 48-year-old man was admitted to the University of Michigan Hospital in March, 1948, with a complaint of hoarseness of six months' duration.

On examination, an ulcerating constructive lesion was visualized on the right side of the epiglottis, aryepiglottic fold, and arytenoid. A histological diagnosis of medullary squamous cell carcinoma was made and the patient received intensive roentgen therapy.

After he was discharged from the hospital, he was not seen again until a year and a half later, when he returned with a complaint of choking on attempting to eat or to drink. He stated that several

From the Department of Otolaryngology, University of Michigan Medical School.

Fig. 1.—Preoperative appearance of the larynx on attempt to phonate.

months after completion of the roentgen treatment three fistulae developed in the right side of the neck, which had drained foul pus and pieces of solid material resembling bone. Undoubtedly he was describing the effects of radionecrosis with secondary suppurative chondritis. Shortly after this occurrence he had developed the difficulty in swallowing, consisting of aspiration of food and fluids. Subsequently, intermittent febrile episodes, chronic cough, and chest pain ensued.

Examination of the larynx revealed absence of the right side of the epiglottis, the aryepiglottic fold, the posterior portion of the ventricular band, and the arytenoid mound, with fixation of the vocal cord in abduction (Fig. 1). Enlarged nodes on the right side of the neck suggested metastatic carcinoma.

Roentgenograms of the chest demonstrated evidence of pneumonitis in the lower lobe of the right lung. There was no clinical or laboratory evidence of tuberculosis.

After establishing positive tissue diagnosis, a right radical neck gland dissection was performed. Postoperatively, the chest condition improved temporarily under antibiotic therapy.

Subsequently, the pneumonitis progressed to a lung abscess, and the patient's general condition became progressively worse. Repeated sputum studies revealed no tubercle bacilli. The patient refused surgical treatment for the abscess, but was persuaded to enter the

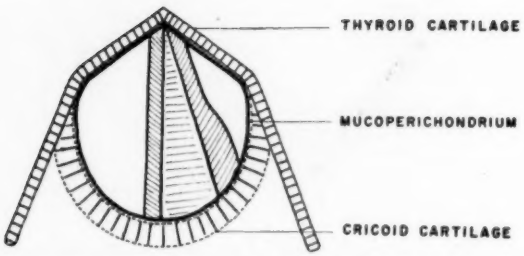

Fig. 2.—Diagram of a cross section through the thyroid cartilage just above the cords.

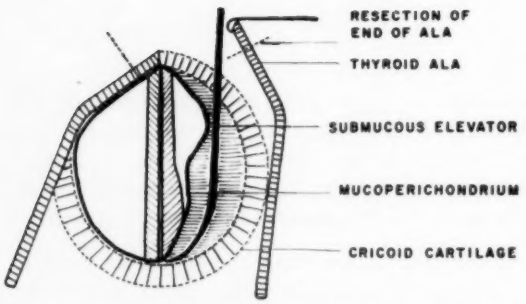

Fig. 3.—Diagram showing elevation of the soft tissues of the lateral wall from the cartilages.

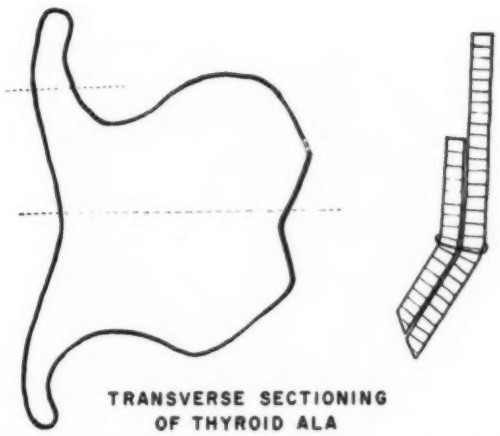

Fig. 4.—Diagram illustrating the portion of the thyroid ala resected for use as a graft.

hospital on two separate occasions for intensive and rather prolonged courses of antibiotic therapy. After each course of therapy he showed some clinical improvement, but repeated chest x-rays demonstrated progressive pneumonitis involving both lungs and enlargement of the abscess cavity.

The patient again was admitted to the hospital and surgical treatment of the pulmonary condition was contemplated. In spite of the proposed treatment, the prognosis was considered poor as long as the patient continued to aspirate food and fluids. It was decided that some attempt must be made to correct the laryngeal deformity, or else to perform a total laryngectomy to effect discontinuity of the alimentary and respiratory passages.

SURGICAL PROCEDURE

After a preliminary tracheotomy under local anesthesia, the thyroid cartilage was split in the mid-line, leaving the mucoperichondrium intact. The latter was elevated from the medial aspect of the right thyroid cartilage to its posterior border. The reflection of the perichondrium around the inferior border of the thyroid cartilage was incised, and the dissection was carried down to the perichondrium of the cricoid cartilage. This, in turn, was incised and elevated from the cricoid cartilage back to the mid-line. In this manner, the soft tissues of the right side of the larynx were entirely separated from

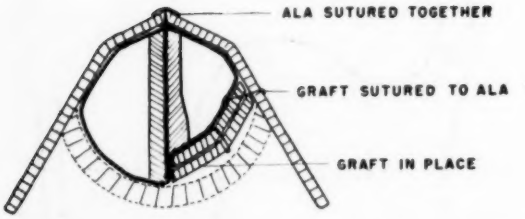

Fig. 5.—Diagram showing the graft in place and the larynx closed.

their rigid support (Figs. 2 and 3). The procedure was accomplished with some difficulty owing to the dense fibrosis and marked adherence of the membranes to the cartilages due to previous x-ray therapy.

In the next step, the perichondrium of the upper half of the left thyroid cartilage was elevated medially and laterally and the upper halves of both thyroid alae were resected and saved for use as a graft. Approximately the anterior centimeter of the remaining portion of each of the thyroid alae was resected to shorten the antero-posterior diameter of the larynx and to mobilize the soft tissues more completely.

The two resected pieces of thyroid cartilage were wired together in double thickness and fashioned into the desired shape as a graft (Fig. 4). This graft was slipped into place between the soft tissue of the larynx and the thyroid and cricoid cartilages. Laryngoscopic inspection demonstrated satisfactory mesial displacement of the right vocal cord. The graft was then wired in place to the remainder of the thyroid ala of that side, and finally the two thyroid alae were wired together anteriorly in the mid-line (Fig. 5). After closing the skin, the interior of the larynx was checked again to ascertain whether the desired effect had been obtained.

The patient had a smooth postoperative course, and remained afebrile. The intensive antibiotic therapy that had been started before the operation, was continued, and he was fed by Levin tube for nine days. The tracheotomy tube was removed on the tenth day. The patient noted immediate improvement in swallowing after the Levin tube had been removed and was able to swallow liquids without aspirating for the first time in nearly two years. His voice, which had been an open whisper, was now strengthened to a hoarse-spoken sound. He was discharged two-and-a-half weeks after operation.

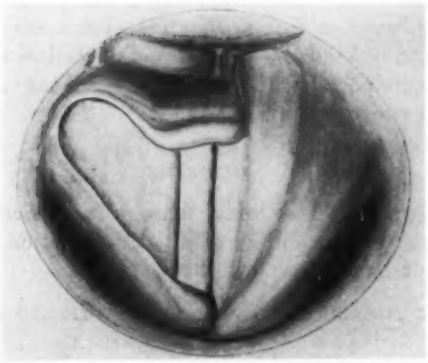

Fig. 6.—Postoperative appearance of the larynx on phonation.

When he returned for check-up examination one month later, he stated that he was swallowing without difficulty and that his voice had gained some strength. Laryngeal examination at that time showed complete disappearance of the operative reaction, and demonstrated complete closure of the glottis on phonation (Fig. 6).

When last examined, two years postoperatively, the patient was able to close the glottis, had a very satisfactory voice, and had had no further trouble with aspiration on swallowing.[2]

COMMENT

The epiglottis serves to deflect food and fluids laterally into the pyriform sinuses, as has been demonstrated by Saunders, Davis, and Miller[1] by means of high-speed cineradiography. The epiglottis, then, is the first line of defense in protecting the lower airway. However, it is well known that an epiglottis can be amputated without causing undue difficulty in swallowing due to aspiration of food and fluids. Likewise, one vocal cord can be completely resected, as is done in the laryngofissure for early carcinoma of the cord, without causing aspiration on swallowing. Many patients having undergone laryngofissure cannot completely close the true glottis, despite the presence of a band of scar tissue that tends to replace the cord. This inability to close the glottis, then, tends to place the burden of functioning on the supraglottic structures; namely, on the false vocal cords and the aryepiglottic folds, which by themselves are able to close the airway during the act of degutition.

In the case just reported, all of the glottic and supraglottic structures were unilaterally involved, being either partially destroyed or distorted by fibrosis so that neither the glottis nor the supraglottic structures could be closed. The destruction and distortion had been caused by a carcinoma, and the radionecrosis and chondritis which occurred after roentgen therapy.

SUMMARY

A case is reported in which chronic pneumonitis and a lung abscess developed as secondary effects of aspiration through a persistently patent glottis.

A plastic procedure that successfuly corrected the deformity is described.

2020 East 93rd Street.

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LXXXV

SUDDEN PERCEPTIVE DEAFNESS

REPORT OF FIVE CASES

MORRIS F. HELLER, M.D.

AND

PAUL LINDENBERG, M.D.

NEW YORK, N. Y.

The occurrence of sudden deafness, monaural or binaural, is not a rare clinical observation. In many instances the etiologies are apparent. Such examples are deafness following fractures of the skull, particularly of the base and pars petrosa; illnesses such as meningitis, measles, or mumps; the use of drugs such as quinine and streptomycin; physical factors such as the bends; and those cases generally described as endolymphatic hydrops. Many times the cause of the sudden deafness is obscure and although there is justification to speculate clinically as to the origin of the deafness, positive proof often is inadequate or absent.

Lehman[1] in his report of two patients who developed sudden deafness referred to the previous writings of Lindsay, Fowler, Schuhknecht, Helmer Rasmussen, de Kleyn, Halsberg and Horton, Loch, Much, Colledge, Hurst et al, Martin, Semenow, Truex, and Hardy. These reports described instances of sudden deafness; in some patients the etiology could be established but in others this could not be determined. Lehman's experience consisted of one patient with deafness of organic genesis and a second in whom psychogenicity played the instrumental role. Saltzman[2] observed patients with fulminating deafness, the etiology of which was otosclerosis.

During 1953 we observed five adult patients who, prior to the abrupt appearance of their deafness, had been well and had no defect in their hearing. These patients offered somewhat similar histories. Each apparently had a cold, rhinitis, or "virus." These infections apparently were not severe, except in the light of the damage to the acoustic and vestibular end-organs.

REPORT OF CASES

CASE 1.—Mrs. N. S., a 42 year old housewife had been living in Morocco. At about the end of December, 1952, she experienced

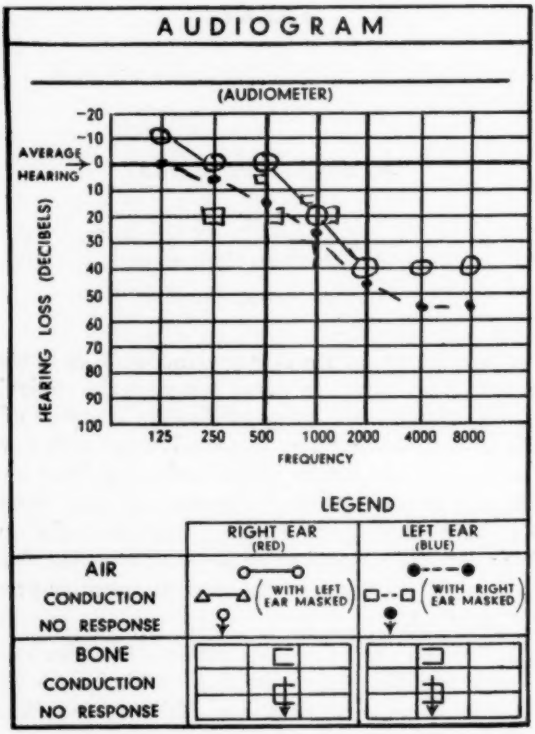

Fig. 1.—N. S., Case 1, May 5, 1953; bone conduction heard only as indicated.

what she described as a very slight cold which did not require any medication for symptomatic relief. Within a few days she recognized that her previous "super acute" hearing was impaired. She described this as though her hearing was muffled. She had no tinnitus or vertigo. There was also some impairment of the sense of smell.

She was examined at the onset of the deafness, and her hearing was tested. A diagnosis of bilateral perceptive deafness was made.

We examined her in May, 1953. Physical inspection of the ears, nose and throat revealed no significant pathology. Functional studies of hearing demonstrated a bilateral perceptive deafness (Fig. 1).

The bone conduction thresholds matched those of air conduction for each ear. By phones the speech reception threshold of the right

ear was 15 dbs, and for the left ear 25 dbs. The reception threshold in the free field was 20 db. The speech discrimination loss for phonetically balanced words at over threshold intensities was about 12 per cent loss.

With a speech reception threshold in the free field of at least 20 dbs, she did not require amplification with a hearing aid. She was advised, however, to take lessons in lip reading.

She returned to us in December, 1953, stating that her hearing loss had increased. Audiometrically her loss was greater for the low tones than originally noted six months earlier (Fig. 2). The speech reception threshold, by phones, for the right ear was now 55 db, and for the left ear 65 dbs. The free field speech reception threshold was 55 db. The speech discrimination loss for the right ear was 26 per cent and for left ear 12 per cent.

A hearing aid was fitted, and at this time amplification was definitely of advantage. The left ear was fitted because of its better discrimination score, and she received instruction in the use of the aid.

CASE 2.—Mrs. W. W., white, 40 years of age, housewife. In June of 1952 she experienced a cold with slight elevation of temperature. On July 4 she left her home with her family and traveled a few miles to a nearby ocean resort. At this time her hearing was unimpaired. Because of her recent infection she did not change into a bathing suit. While seated at the beach her family noticed she did not reply when they addressed her. She had no vertigo. Eventually she noticed the onset of tinnitus, which became most annoying and from which she sought relief. Her past history included an enlarged thyroid gland, with a lowered basal metabolic rate. Examination September 12, 1953, of the ears revealed both tympanic membranes to be thickened, scarred, retracted, with limited mobility. The deviation of the nasal septum was not pathological. The thyroid gland was somewhat enlarged. Audiometric studies revealed a bilateral perceptive deafness, the right ear being somewhat better than the left (Fig. 3). The speech reception threshold, by phone, for the right ear was 45 db, and for the left ear 55 db. The speech discrimmination loss for the right ear, at over threshold intensities, was 16 per cent and for the left ear 46 per cent. The binaural speech reception threshold in the free field was 45 db.

A trial of nicotinic acid orally produced generalized flushing, but no acoustic changes, nor was the tinnitus altered.

A hearing aid fitting was attempted, but she received no improvement with amplification. Her tolerance to increased intensities was poor. She was advised to take lip reading instructions.

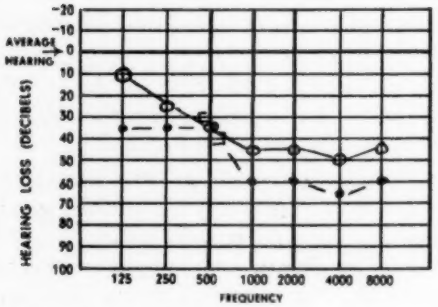

Fig. 2.—N. S., Case 1, December 8, 1953; bone conduction heard only as indicated.

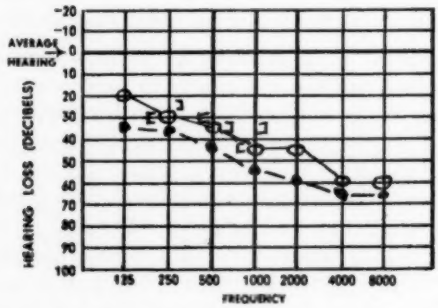

Fig. 3.—W. W., Case 2, September 12, 1953; bone conduction heard only as indicated.

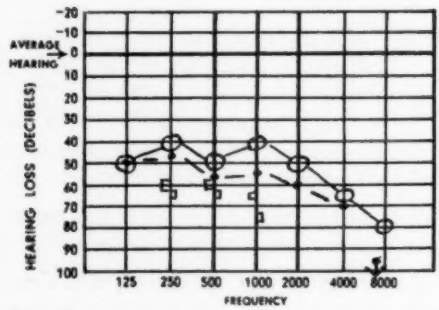

Fig. 4.—M. R., Case 3, December, 1953, bone conduction heard only as indicated.

CASE 3.—(Courtesy of S. Evans Ganz, M.D.): Mr. M. R., aged 27, white male. In August, 1953, he experienced what he described as "virus." His sense of balance, he said, was impaired, and he noticed head noises and a sudden development of marked deafness bilaterally. He also had at the onset an acute conjunctivitis and photophobia. There was no apparent elevation in temperature. He was given cortisone as well as other medication, none of which influenced the deafness. He was first examined by Dr. Ganz in December, 1953, at which time the physical examination of the ear, nose and throat revealed no pathology. There was no vertigo or nystagmus. The audiogram demonstrated bilateral perceptive deafness (Fig. 4).

CASE 4.—Mrs. P. L., aged 30, white housewife, noticed the onset of deafness in the left ear on about October 5, 1953. This had been preceded by a slight rhinorrhea of a few hours duration. Her own voice and other voices sounded as an echo. Left tinnitus was present. She had a slight dizzy-"swimming," sensation. She described some deep pain in the region of the left mastoid process, "like a bone bruise." The patient, incidentally, has recurring allergic symptoms, usually relieved by medication. Seven days after the onset of deafness, she consulted us. Physical examination on October 12, 1953, showed the ears, nose and throat to be essentially normal in appearance. There was no corneal anesthesia. Her two-point discrimination was intact. There was some nystagmus, which was marked on right gaze. However, the patient has a squint. Consultation with her ophthmalogist determined subsequently that there was no significant nystagmus. There was hyperesthesia to pin prick of the skin over the left mastoid process and around the left auricle. This area seemed to correspond to the sensory neural distribution of the auricular-temporal branch of the mandibular division of the fifth cranial nerve and to the great auricular branch of the second and third cranial nerves. Caloric vestibular tests of each ear were performed, but these were inconclusive in eliciting nystagmus. Neurologically, the biceps, deep knee and ankle reflexes were equal and active. There was slight tightness of the neck and a suggestion of a bilateral Kernig. The right abdominal reflexes were less active than the left. (An appendectomy had been performed the year before.)

At the time of her first visit to us, the following hearing tests results were noted; pure tone audiometry demonstrated normal hearing for the right ear. The pure tone air conduction for the left ear without masking or with masking of 70 db demonstrated thresholds of 30 db. However, with 80 db of masking, the following thresholds were noted: For the frequencies of 125 cps 30 db, 250 cps 20

db, 500 cps 30 db, 1000 cps 70 db. No thresholds were obtained at 2000 cps, 4000 cps, 8000 cps. The bone conduction thresholds with sufficient masking demonstrated no response at 2000 cps and at 4000 cps. At the lower frequencies the bone conduction thresholds were similar to the air conduction thresholds (Fig. 5).

The speech reception threshold for the right ear was 5 db by phones with no loss for discrimination. The speech reception threshold for the left ear by phones with masking was 35 db and at over-threshold intensities the speech discrimination loss was 40 per cent.

She was re-examined the following week. The painful feeling in the region of the left mastoid process had abated somewhat. Her sensations of dizziness were only momentary and infrequent. She noted asteriophonia, all environmental sounds seemed to be on the right side. There was no tinnitus. Voices still echoed. She described the pain as being very deep in her head. She also said that low tone noises bothered her. The physical examination at this time revealed no significant pathology except that the posterior cervical nodes of the left neck were shotty and moderately tender. The equivocal nystagmus was still present. The hyperesthesia was still present about the left preauricular area.

When seen again on May 1, 1954 she supplied the following information: There was a gradual improvement in the hearing of the left ear to which she thought was normal hearing. In January, 1954, there was a recurrence of sudden deafness of the left ear, not as severe as that of the original episode, which after one week recovered again.

Re-tests of the left ear with masking of the unaffected normal right ear, demonstrated normal hearing throughout the auditory spectrum (Fig. 6). The pure tone thresholds of the left ear were raised slightly when compared with the right, when the right ear was masked. The spectrum of the left ear without masking varied between 0 db and 10 db.

CASE 5.—Mr. M. F., aged 27, in August, 1953, developed a "virus" infection with a temperature to 101° F. A few weeks later he noted conjunctivitis and photophobia. He felt a puffiness of his ears, bilateral deafness, tinnitus, vomiting, nausea and vertigo. The vertigo was so severe that for a few days he staggered and was unable to walk without holding to supports.

We saw the patient for the first time on December 29, 1953.

The ear, nose and throat examination was entirely negative. There was no nystagmus. The Romberg was negative, but he staggered when walking with his eyes closed.

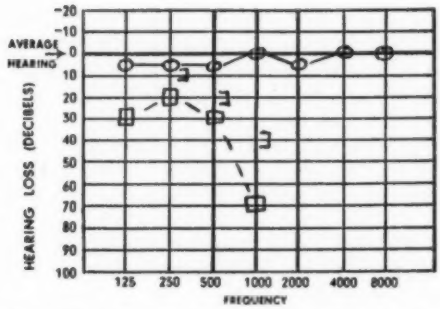

Fig. 5.—Mrs. P. L., Case 4, October 14, 1953, left ear air and bone conduction heard only as indicated.

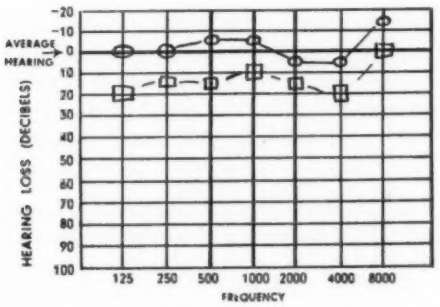

Fig. 6.—Mrs. P. L., Case 4, May 25, 1954, recovery of hearing of left ear.

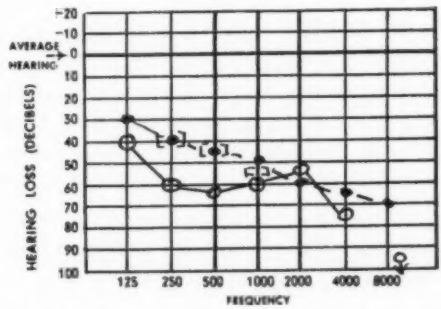

Fig. 7.—M. F., Case 5, December 29, 1953, bone conduction only as indicated.

Pure tone audiometry demonstrated a bilateral perceptive deafness (Fig. 7). The right speech reception threshold was 65 db, with a PB loss of 52 per cent. The left speech reception threshold was 49 db, and the PB loss was 16 per cent. The free field binaural speech reception threshold was 38 db with a PB loss of 14 per cent.

Each statokinetic end organ was stimulated with 50 cc of ice water. No nystagmus was observable, nor did the patient subjectively experience any sensation of discomfort or dysequilibrium.

He was fitted with a hearing aid, obtaining a speech reception threshold of 19 db with the instrument worn in the left ear. There was no loss or gain in discrimination with the aid.

He returned again on March 29, 1954, complaining of increasing deafness of the right ear. This was confirmed by audiometry (Fig. 8).

DISCUSSION

The history, physical and functional findings of five adult patients who suddenly became deaf has been presented. Three of the patients were females, two were males. All had been apparently in good health and none suffered from any debilitating or chronic diseases.

One patient developed monaural deafness, spontaneously recovering normal hearing with normal audiometric levels.

The four patients who became bilaterally deafened seemed to have sustained approximately the same depth of loss audiometrically. Their speech reception thresholds were in agreement with the audiometric pure tone thresholds for air conduction. The discrimination losses for phonetically balanced words varied amongst the patients, and also differed between pairs of ears.

Two patients (Cases 1 and 5) demonstrated over a period of time a further loss of hearing, greater than had been noted at our first examination. These patients were examined originally several months after the onset of the deafness, at a time when it might have been estimated that the loss had become stabilized. Nevertheless, subsequently they experienced additional hearing loss.

One patient (Case 2) complained of severe tinnitus which prompted her to seek relief. The other patients did not consider tinnitus as a prominent or disturbing symptom.

Two patients, both males (Cases 3 and 5) had at the onset of their illness most severe vertigo, dysequilibrium, nausea, vomiting—

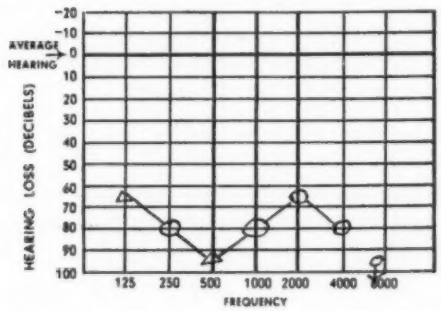

Fig. 8.—M. F., Case 5, right ear only, no bone conduction heard, left ear, not depicted, no change; see Fig. 7.

and for several days were unable to perambulate because of their loss of stability. Case 5 eventually demonstrated a loss of function of the statokinetic end organs as evaluated by cold caloric stimulation. In addition both patients had a conjunctivitis which in Case 5 seemed to have become chronic. Case 4 had experienced slight dizziness which was not at all incapacitating or annoying.

In the history of these patients there is a suggestion, at least, that the deafness was preceded by what appeared to them to be a rhinitis, not necessarily severe. These were not characteristic upper respiratory tract infections. Can one proceed from here to speculate upon the mechanism which might have produced the deafness?

Allergy, in the usual sense, is not in evidence in this group. Case 4 has a history of allergic disease, and was the only patient to recover. Her deafness was monaural, which may be evidence that this was not an allergic otic episode. Nevertheless, her recovery may be indicative of reversibility of allergic disease.

A second possible mechanism is that of blood sludging. Sludging has been reported as a phenomenon associated with several pathologic states. It follows, then, that cause of the sludging must be determined, and it is presumed that the vascular system of each inner ear was so implicated.

Another possible consideration is that they were infected with a virus which has an affinity for neural tissue, and that in these patients, their otic system was the recipient of the viral insult, rather than some other neural structures.

It is understood that these patients may not have become deafened by an agency common to all, and that each had become deafened by a different etiological agent. This certainly would increase the difficulties in uncovering the cause of deafness in such patients, and in clarifying a single clinical picture.

Another question intrudes itself: Why, in the four patients with bilateral perceptive deafness has the hearing receded to similar audiometric levels and configuration; and furthermore, why do the discrimination losses vary? The testing procedures, we think, are not responsible for the variations.

It would seem that some answers to the etiology might ultimately be found if patients are seen at the onset of their sudden perceptive deafness. Studies of the spinal fluid and blood may reveal the presence of viruses or biologic responses compatible with invasion of such organisms.

Presumably sudden perceptive deafness can occur at any age. Obviously, to become aware of the deafness and complain about it, the patient must be old enough to differentiate and communicate. This is denied the infant, and it can be conjectured that many instances of so-called inherited deafness are in fact the result of similar otic damage. This does not preclude the occurrence of such damage prenatally.

SUMMARY

1. Five adult patients have been presented who developed sudden perceptive deafness.

2. Four patients developed bilateral deafness which apparently is irreversible.

3. One patient developed monaural deafness and recovered spontaneously.

4. No etiological factors have been identified, but hypotheses are offered.

Cases 1, 2, 3, 5 were tested in sound treated rooms, the ambient noise levels of which are less than 30 db.

115 East 61st St.

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LXXXVI

MÉNIÈRE'S DISEASE: SURGICAL TREATMENT

HENRY M. GOODYEAR, M.D.

CINCINNATI, O.

In the March issue of the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY Dr. J. R. Lindsay and Dr. K. H. Siedentop[1] report on "Labyrinthine Surgery in the Treatment of Ménière's Disease." They state "Goodyear[2] reported relief from vertigo by making a fistula in the horizontal canal and puncturing the membranous labyrinth (utricle) with a barbed dental broach."

Perhaps this was an oversight on the authors' part for I have never mentioned puncture of the utricle in any procedure I have described in operation for Ménière's, nor have I ever used a barbed dental broach.

It has been my experience that upon entering the vestibule through an opening in the anterior end of the horizontal canal the entire contents of the vestibule can be completely distorted and destroyed by use of a dental explorer or endosteal pick.

The endolymphatic membrane is very delicate and loosely hung in the bony cavity of the vestibule. The most important islands of neuro-epithelium are in the ampulla and the adjacent portion of the vestibule as a part of the membrane constituting the utricle.

In 1943[3] I first reported the destruction of the utricle in extreme cases of Ménière's. No idea of a simple puncture was conveyed in this article.

For many years I have supervised the dissection and destruction of forty adult labyrinths or more per year, plus as many labyrinths in infant heads. The vestibule is very small and any small instrument which can be moved freely about in the cavity whether through an opening in the anterior end of the horizontal canal or any other part of the vestibule will completely destroy the utricle and saccule.

I have destroyed the utricle and saccule routinely in my operations for Ménière's syndrome, entering the vestibule through an opening in the anterior end of the horizontal canal. In not a single instance has there been any residual function of either hearing or vertigo in any of these cases following operation.

I disagree that it is necessary to make a broad opening connecting both the anterior ends of the horizontal and superior canals in order to destroy this very delicate membrane in the vestibule. I also disagree that it is necessary to remove any portion of the membrane from the vestibule. My experience both in the anatomical laboratory and in actual operations bears me out in this conclusion.

I am sorry that so much ado is made regarding the destruction of the contents of the vestibule. One might think it was necessary to remove this delicate endolymphatic membrane and take it well away for fear it might regenerate and produce a recurrence of vertigo. It is so easy to completely destroy this membrane, once the vestibule is open, that there should be no controversy.

DOCTORS BLDG.

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Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, October 11, 1954

A joint meeting of the Chicago Laryngological and Otological Society and the Chicago Ophthalmological Society was held at the Drake Hotel.

Nasolacrimal Surgery

J. VERNAL CASSADY, M.D.

SOUTH BEND, IND.

AND

GORDON H. SCOTT, M.D.

CHICAGO, ILL.

(Abstract)

DR. J. VERNAL CASSADY: Since ophthalmology and otolaryngology have separated, the distance between them anatomically is not the same distance that it was when both were practiced by the identical physican. Such a combined meeting as this helps us to take stock of the other's approach to similar problems. Unity and friendship so essential to the dignity and usefulness of our profession is cemented by this type of combined association and is just as important as is its educational value.

Before rushing in to treat lacrimal drainage obstruction, we should be sure that such a problem exists and that it is not simply a matter of excessive lacrimation. When we investigate lacrimal drainage, the position of the punctum and the presence of allergic rhinitis or sinus disease should be considered before exploring an obstruction in the lacrimal duct which may be functional or secondary.

If lacrimal duct surgery is necessary, the Dupuy Dutemps method of dacryocystorhinostomy is advocated and used by most ophthalmologists. Unless the new nasal opening is lined with mucous membrane

it often will not remain patent but tends to scar over and cause recurrence of the obstruction. Some refinements of the original Dupuy Dutemps surgery are pointed out, the use of the Strycker saw for trephining into the nose and the use of small curved D and G No. 269 dacryocystorhinostomy suture needles instead of the Reverdin needles originally used. The importance of a large new opening into the nose, the removal of the lacrimal spur at the lower end of the anterior lacrimal crest, a method of incising the nasal mucosa to facilitate its suturing is depicted in the film presentation.

Dr. Gordon H. Scott: Sixty-three cases of chronic dacryocystitis were operated upon intranasally by the West technique. Good results were obtained in 58 cases; fair in one and poor in four.

The ostium should be made by removal of portions of the frontal process of the maxilla and of the lacrimal bone. The opening should be adjacent to the root of the middle turbinate and high enough to expose the cupola of the sac. Complete removal of the medial wall of the sac ensures clearance of the orifices of the lacrimal canals which enter the sac in its upper third.

Failures could be attributed to inadequate bone removal or a narrowing of the lumen in the lacrimal canals. One case was relieved by inserting plastic tubing in the inferior canal for four days. Had this material been available earlier there might have been fewer failures.

DISCUSSION

Dr. Maurice D. Pearlman: I thought it might be of interest to tell you that during February of this year, Dr. Iliff of Baltimore presented a technique in which he used a soft rubber catheter, attached to the lacrimal sac and coming out through the nostril, to maintain the opening and to assure close apposition of the sac to the nasal mucosa. We have tried this on a few cases at the Infirmary and are disappointed because the soft rubber catheter apparently induces hyperplastic scar formation and unusual swelling in the operative site. Later, retraction of the caruncle occurred which was disfiguring. I thought it might be of interest to report these experiences.

Dr. J. Vernal Cassady (closing): The method referred to by Dr. Pearlman is similar, of course, to that of Stoltz and Gifford, in which they brought the end of the sac through the trephine opening with sutures down into the nose. We tried this, but failed to find that it would stay open. Dr. Gifford was still enthusiastic about it when I talked to him in New York.

Dr. Lester Jones sent a questionnaire to a large number of medical centers asking what method of dacryocystorhinostomy was taught to their residents. Thirty-five per cent were done by the method I showed, the Dupuy Dutemps. Then another 25 per cent were done by the method of Mosher and Arruga. He asked whether they had rhinological examination before surgery and 95 per cent of answers were in the affirmative. But the external method of dacryocystorhinostomy was used in 95 per cent of the cases. I think the reason for this is that more of these cases come first to ophthalmologists rather than to otolaryngologists.

(Slide) This is a section of a full term stillborn infant, showing the nasolacrimal duct under the inferior turbinate, which I think shows how easy it is to have obstruction in infants. In the ducts I examined anatomically at the Carnegie Institute, 85 per cent were closed a birth. In these cases there is a membrane over the lower end of the duct and it is important to open this up so that dacryocystitis does not develop later.

Related Lesions of the Orbit and Nasal Sinuses

JACK P. COWEN, M.D.

LINDEN J. WALLNER, M.D.

AND

STANTON A. FRIEDBERG, M.D.

CHICAGO, ILL.

DR. JACK P. COWAN: Of the affections of the orbit, those producing asymmetric exophthalmos more frequently are the following: exophthalmic goiter, either of the thyrotoxic or the thyrotropic variety; then hemangiomata, chronic orbital granulomata or pseudotumors, mucoceles and aneurysms; meningiomata, extension of malignancy of other paranasal sinuses and primary malignant tumors of the orbit are next in importance; lastly, benign primary orbital tumors, dermoids and mixed tumors of the lacrimal gland and lymphomata.

Asymmetric exophthalmos in a large series of cases was found to be due equally to neoplastic and non-neoplastic causes. Of the neoplastic orbital lesions producing exophthalmos, half were benign in origin, half were malignant. Seventy-five per cent of benign tumors of the orbit produce exophthalmos. Of the malignant variety, they were equally divided into those arising within the orbit and

those entering the orbit secondarily; the latter will show x-ray evidence of involvement in 90 per cent of cases.

DR. LINDEN J. WALLNER: The pathogenesis of inflammatory lesions of the orbit of nasal origin is explained by the anatomy of this region. The orbit is described as a four-sided pyramid, surrounded on three sides by nasal air sinuses. The frontal sinus forms the roof, the sphenoid and ethmoid form the medial wall and the maxillary sinus forms the floor. The antrum is the most frequently infected of the sinuses, but it rarely leads to orbital involvement. Infection of the ethmoid sinus is the most common cause of orbital cellulitis in children, frontal sinusitis is the most frequent source in adults.

The superior and inferior ophthalmic veins drain the area supplied by the ophthalmic artery. The superior ophthalmic vein receives the anterior and posterior ethmoidal veins, and has a large communication with the facial system by means of the angular vein. The inferior ophthalmic vein communicates with the pterygoid plexus. The two ophthalmic veins do not follow the artery out of the orbit, but leave through the superior orbital fissure to enter the anterior wall of the cavernous sinus. There are no lymphatic vessels or nodes in the orbit.

There has been little opportunity to determine the exact pathology of orbital cellulitis since antimicrobial therapy has been available. Occurrence of the disease is rare compared with former years and it responds readily to chemotherapy. Even before 1938, when surgical exploration was performed frequently, there was disagreement as to the pathology. A subperiosteal abscess was often found, indicating that the correct terminology should be orbital abscess. The term cellulitis indicates a diffuse infection of the soft tissues of the orbit. This has been described but is probably rare. Orbital cellulitis or abscess and cavernous sinus thrombosis present edema and discoloration of the lids, proptosis and limitation of motion of the globe, and chemosis. These symptoms may all be explained by venous obstruction. This alteration of venous drainage would seem to be the common denominator, producing similar symptoms in diseases with widely different types of pathology.

Kodachrome transparencies were shown of a group of illustrative cases of orbital cellulitis and abscess in children and adults, mucocele, osteomyelitis of the frontal bone, early orbital involvement from folliculitis of the vestibule, cavernous sinus thrombosis, and trauma of the orbit. The diagnosis and treatment were discussed briefly.

DR. STANTON A. FRIEDBERG: Large dentigerous cysts must be included among the lesions which may produce orbital complications.

In this instance (slide) the cyst had caused destruction of the walls of the maxillary sinus and erosion of the infra-orbital plate. As with mucoceles (slide) these cystic masses are likely to attain considerable size without causing pain, unless secondary infection supervenes.

In the group of benign tumors of the nasal passages and sinuses we often find that damage to adjacent structures caused by expansile growth is far more serious than one might expect from the histologic picture. Among the examples shown to illustrate this point are vascular fibroma of the nasopharynx (juvenile nasopharyngeal fibroma) which had invaded the orbit via the inferior orbital fissure, a huge osteoma which occupied the greater portion of the right orbit and ethmoid region, and a chondroma arising in the nasal septum and invading all the sinuses. This benign giant cell tumor of the sphenoid caused slight proptosis and diplopia. Chordomas of the sphenoid area may invade the orbit and nasal passages, as borne out by these slides of an illustrative case. Pituitary adenomas have been shown to extend intranasally via the sphenoid sinus in two patients described several years ago.

In the group of malignant tumors of the nose and sinuses, extension to the orbit often occurs in the usual course of the disease. Removal of the maxilla, sometimes with the contents of the orbit, may offer the best possibility of cure. Examples of these procedures were shown. Irradiation therapy is often successful in controlling localized lesions of the lymphoma group of tumors, but development of metastases or distant foci of disease may be anticipated.

Meeting of Monday, November 1, 1954

THE PRESIDENT, DR. NORMAN LESHIN, IN THE CHAIR

The Surgical Anatomy of the Facial Nerve and Parotid Gland

BARRY J. ANSON, PH.D.

AND

RICHARD A. DAVIS, M.D.

CHICAGO, ILL.

(Abstract)

The parotid gland together with its duct and the facial nerve were studied in dissections of 300 cervicofacial halves in order to provide additional information on the anatomy of each of these structures separately and to record the major aspects of their interrelationship.

The size of the gland is a highly variable feature; the vertical dimension (height) was found to vary between 9 cm and 6 cm as extremes. In horizontal plane (width) the extremes were 5 cm and 2 cm.

Relationships of the gland vary, of course, with its dimensions. But in addition to differences which depend upon the extent of the area covered, heterogeneity in arterial and venous supply make for further complexity. Related skeletal structures are also variable. The styloid process belongs in this category. Frequently absent or very short, the process occasionally attains a length of 3.6 cm.

Despite considerable variation in size, shape and relationships, the parotid gland is invariably bilobed; the portions, superficial and deep, are connected by an isthmus. Between these leaves course the branches of the facial nerve and certain of the blood vessels which supply the face. Additionally, the "ductules" which join to form the single parotid duct of each side pass through this same interlaminar space.

Quite regularly the trunk of the facial nerve separates proximally into divisions, termed cervicofacial and temporofacial. In more than 80 per cent of the cases these divisions, or the adjacent members of the set of five derived nerves, anastomose to form a plexus in any one of eight or more patterns. Intricate schemes heavily outnumber the simple. This plexiform set of anastomoses is likely to occur in the space between the leaves of the gland, the nerves then emerging from beneath the anterior margin of the superficial part.

The fascial investment of the parotid gland is strong externally, where the layer contains fascicles of the muscles of facial expression. Internally the gland is related to the deep fascia of the masseter. These layers do not enclose the gland tightly, and hence do not form a capsule in the usual meaning of the term.

DISCUSSION

DR. PIERCE THEOBALD: I am sure we all owe a great debt of gratitude to Dr. Anson for his usual masterly presentation. Those of us who approach surgery of the parotid gland with timidity will now have a more firm footing which will be helpful to us all. Although our timidity will not be decreased, we may approach the gland with more confidence than heretofore.

DR. M. F. SNITMAN: Those of us who are familiar with the anatomy of the parotid gland can appreciate the labor involved in the dissection of this large number of glands. In our experience I do not recall ever having seen a parotid tumor that was not super-

ficial to the facial nerve and therefore in the superficial part of the gland. Is there perhaps some embryological reason for this?

Dr. Thomas C. Galloway: I want to pay tribute to this beautiful anatomical study. I have had a somewhat different approach to parotid surgery from some operators. I have thought that by very closely following the intact capsule of the tumor rather than first exposing the nerve, I have had fairly good success in removing such neoplasms. We hear about complete removal of the parotid gland, and I would like to ask Dr. Anson if that is surgically possible. Some advocate piecemeal removal, emphasizing that the nerve be left intact. It is my experience that the capsule generally pushes aside the facial nerve in cases which are at all favorable.

Developmentally and anatomically, is there any difference in the structure of the deeper portion of the gland, which makes it less likely to develop so-called mixed tumor of the parotid gland?

Dr. Burton Soboroff: I should like to ask one question which, although related to the anatomy of the gland, may be complicated by the presence of a tumor. There has been some question as to whether it is best to approach the main stem of the nerve or best to find a peripheral branch and follow it back. I have felt it was best to try to find a branch and trace it back to the main stem, feeling that this is safer. I should like to ask whether it is safe from an anatomical standpoint to follow one branch and, if so, which might be best to follow to the main stem.

Dr. Jack A. Weiss: I should like to ask whether in these multilooped nerve branches one can be sacrificed without loss of innervation to the muscle it serves. Does a fractional loss ever occur, without losing the response of the muscle as a whole?

Dr. Barry J. Anson (closing): When I mentioned that a capsule (in the ordinary meaning of that term) was wanting in the case of the parotid gland, I think that statement was over-strong. The gland is invested by fasciae, but is not tightly covered as are, for example, the liver and the spleen. An outer layer contains the musculature of facial expression, while an inner stratum covers the muscles of mastication; the gland is lodged rather loosely between them.

I am baffled by the reported observation that tumors are more likely to occur in the superficial portion of the gland than in the deep. I can offer no explanation for this odd predilection.

Admittedly, reports on normal anatomy do not provide the surgeon with all the information needed to solve a clinical problem.

Unfortunately, we have had no opportunity to study facial nerves distorted by parotid tumors. We should conduct such research as a sequel to the current phase of the investigation.

Regarding the matter of overlapping innervation, I am of the opinion that supply would be furnished through the plexiform arrangement of the various branches of the facial nerve. Specifically, were fibers from a temporal ramus severed there would be, in many instances, an auxiliary supply from a connecting branch of zygomatic source.

Based on anatomic experience, I would say that the facial nerve (for freeing of its branches) should be approached at its proximal portion, that is, near the point where it emerges from the stylomastoid foramen; attempts to identify and follow the small peripheral twigs may end in failure or, if successful, may require needlessly tedious effort.

Congenital Anomalies of the Larynx

PAUL H. HOLINGER, M.D.

KENNETH C. JOHNSTON, M.D.

AND

FILMORE SCHILLER, M.D.

CHICAGO, ILL.

(Abstract)

Statistical analyses of four groups of congenital laryngeal anomalies were presented: (a) congenital laryngeal stridor; (b) webs and atresia; (c) subglottic stenoses and, (d) cysts and laryngoceles.

Of 305 infants having the typical, flaccid larynx recognized as congenital laryngeal stridor, 199 were males, 106 females. The tubular epiglottis, fluttering arytenoids sucked into the glottis on inspiration and the not infrequently associated funnel breast were of note. Nineteen infants, nine males and ten females, have been examined who have had congenital laryngeal webs. Thirteen had a tough, arched membrane involving the anterior one-fourth to seven-eighths of the glottis; one was found to have a transparent sheet across the superior surface of the cords; in two the web appeared to be a fusion of the anterior two-thirds of the cord surfaces, and in three cases a

supraglottic fusion of the anterior half of the false cords and aryepiglottic folds was found lying over the normal true cords. One infant, tracheotomized immediately after birth, had a complete laryngeal atresia. There were 34 infants, 20 miles, 14 females, with congenital subglottic stenosis; in two a gross deformity of the cricoid cartilage was responsible for the obstruction. Congenital laryngeal cysts have been observed in five newborn infants; three originated in the right laryngeal ventricle to cover the true cord and obstruct the glottis, and two originated in the left arytenoid and aryepiglottic fold, severely compressing the glottis. Fifteen adult patients with laryngoceles are mentioned in this category as having possible developmental anomalies; in 11 cases the laryngoceles were unilateral, in four, bilateral.

Symptoms of most of the congenital laryngeal anomalies indicated laryngeal disease, but were not characteristic of any specific lesion. Most infants had symptoms of stridor, dyspnea or cyanosis associated with a hoarse, weak or absent cry from birth until examination. Roentgen studies included neck and chest films and fluoroscopic examination of the esophagus. Diagnosis was established by direct laryngoscopy, but in questionable cases a bronchoscope was passed between the cords and into the trachea to determine the level at which obstructive signs and symptoms were relieved as the scope was advanced. Treatment for the most part was conservative, with tracheotomy dependent on the symptoms of obstruction rather than upon the type of anomaly.

DISCUSSION

DR. THOMAS C. GALLOWAY: I would like to ask Dr. Holinger if he has used the scheme of Iglauer in congenital lesions. He used a device like a jeweler's ring, which is put on after splitting the web but leaving the margin until epithelization occurs. Tantalum has been advised for similar use. This is done intralaryngeally.

DR. PAUL H. HOLINGER (closing): We have not used the ring Dr. Galloway mentioned. It is a technique which consists of placing a ring in the anterior part of the web and leaving it so that epithelization occurs before the web is cut. It would seem that there is some danger of the ring being aspirated, and there may be danger of infection.

I did not mean to indicate that toxins alone are responsible for congenital anomalies, but certainly they are for some of them; as, for example, the toxins associated with German measles in the first trimester, often resulting in many congenital anomalies. This is one that may be effective.

Meeting of Monday, December 6, 1954

THE PRESIDENT, DR. NORMAN LESHIN, IN THE CHAIR

Giant Cell Lesion with Brachial Plexus Symptoms: Case Report

ARTHUR L. RATKO, M.D. (By Invitation)

This case is presented because of two unusual features: The progressive symptomatology was justifiably attributed to a concomitant disease process, and giant cell tumors of the cervical vertebrae are rather uncommon.

The patient was a 19 year old white female, first admitted to the Research and Educational Hospital with a tentative diagnosis of lupus erythematosus disseminatus on September 19, 1951. She had the typical "butterfly" erythema of the face and complaints of fever, anorexia, and marked fatigue. Bone marrow biopsy confirmed the diagnosis. Intravenous ACTH was administered; the symptoms rapidly subsided and she was discharged from the hospital on November 1, 1951. Thereafter the disease process was controlled by administration of vitamins, testosterone, cortone, and occasionally antibiotics. She was hospitalized in March and again in September, 1952, because of flareups, but each time intravenous ACTH produced a prompt remission.

In March, 1952, for the first time the patient complained of severe intermittent pain, numbness and tingling in the left arm and shoulder, occurring in attacks lasting five to ten minutes several times daily and leaving a persistent dull ache in the left shoulder. Physical examination disclosed no significant findings. A chest x-ray disclosed an enlarged left cardiac border and a hazy hemidiaphragm. During the next year the symptoms became progressively more severe and, although there was some response to intensified cortone therapy, she was never free from pain and numbness.

On March 16, 1954, the patient was referred to the ear, nose and throat clinic for evaluation of a "lump" in the left neck and slight dysphagia of about three months' duration. A firm smooth mass, approximately 2.5 cm in diameter, was found under the lower third of the left sternomastoid muscle; the mass was slightly movable. There was slight bulging of the left posterolateral wall of the hypopharynx. An aspiration biopsy of the mass was done, as well as barium study of the esophagus and cervical spine and chest films. The barium study and chest films were essentially normal. The spine

films, however, disclosed a punched-out defect of C-6, a "washed-out" C-5 and C-7, and absence of intervertebral disks between these vertebrae. The aspiration biopsy was reported as "tiny fragments of fibrinous material containing sheets of large, atypical multinucleated cells" with a tentative diagnosis of "malignant appearing cells." Further biopsy under local anesthesia on April 5, 1954, disclosed the mass deep to the posterior border of the lower third of the sternomastoid muscle, measuring approximately 3.0 by 3.5 cm. It was pale and hard, and there was marked fibrosis with some distortion of the surrounding structures. It was felt that total excision was not feasible and a wedge biopsy was taken. The histopathologic diagnosis was "invasive giant cell tumor, probably originating from C-6."

Since lesions similar to this one are occasionally seen in the presence of parathyroid dysfunction, this probability was ruled out by appropriate laboratory studies. All previous x-rays were carefully reviewed. Surprisingly, a punched-out defect was clearly demonstrated in C-6 on the chest x-ray taken the first day the patient complained of pain, numbness and tingling. The slowly enlarging defect was then noted on chest films taken prior to her first visit to our clinic. Two salient factors in establishing the malignancy of a giant cell tumor are: a) Clearly malignant histopathologic characteristics of the stromal cells, and b) their rapidly progressive clinical course. Since neither criterion of malignancy was present the decision was made to treat the lesion as a benign tumor. The possibility of cord damage, if surgical excision and bone graft were attempted precluded this therapeutic approach. Accordingly, a calculated tumor dose of 1090 r was delivered to the lesion via right and left lateral ports. A removable semirigid collar was prescribed to stabilize the spine, since further decalcification of the vertebrae was anticipated. A severe weeping erythema of the neck developed, which was attributed to the abnormal skin sensitivity in the presence of lupus. The reaction subsided rapidly following completion of therapy.

Repeated cervical spine films demonstrated the anticipated decalcification for a period of approximately two months with no apparent change in the size of the bony defect. The mass in the neck, the hypopharyngeal swelling, and symptoms of pain and numbness gradually subsided simultaneously. A second course of radiation was delivered with a calculated tumor dose of 800 r from October 18, 1954 to October 27, 1954. Films taken on October 18, 1954, showed increased sclerosis which is probably indicative of increased calcification within the lesion. The patient was last seen on October 28, 1954, and had no complaints of pain or numbness in the shoulder or arm, but had tingling if she wrote for excessive periods. The mass in the

neck and the hypopharyngeal swelling have disappeared and she has gained approximately ten pounds, is working regularly as a secretary, and appears to be well.

DISCUSSION

DR. JOSEPH G. SCHOOLMAN: We have all had occasions when concentration upon one disease entity has obscured the picture of other things that may be present. In one dramatic incident which I published some years ago, a lymphoblastoma originating in a tonsil was overlooked, or at least the diagnosis was put off, because of concentration upon the original pathology in that patient. We should keep in mind that, despite our original teaching we should always look for one answer to all pathology, all cases do not fit that rule, and often more than one entity may be present.

DR. ARTHUR L. RATKO (closing): Before the decision as to treatment was made there was quite a discussion. The orthopedic department recommended block excision and bone graft. Here we had three vertebrae almost completely destroyed; the brachial plexus, jugular vein and posterior wall of the hypopharynx were matted to this mass. How one could possibly stabilize the spine after an excision of such magnitude was more than we could see. Further, the radical type of surgical procedure is usually reserved for the malignant type of lesion, and we felt that this was benign. One of the arguments against radiation therapy is that frequently osteogenic sarcoma or malignant giant cell tumors have arisen in previously irradiated benign giant cell tumors. One wonders, in going through the literature and noting series of cases in which little or no therapy, or even surgical therapy, was instituted, and malignant tumors have developed at the site of a previously benign tumor, how radiation therapy can be indicated. The consensus of opinion is, however, that the choice of therapy depends upon the location and character of the lesion.

End-Organ Deafness

KINSEY M. SIMONTON, M.D. (By Invitation)

ROCHESTER, MINN.

(Abstract)

The concept of end-organ deafness as a distinct classification was elaborated by Mygind in 1947. Various characteristics of cochlear deafness had previously been noted by Ménière (1861), Shambaugh

(1920), and Fowler (1920). The pathologic basis for the syndrome is thought to be disturbance of the hair cells in Corti's organ. Endolymphatic hydrops is assumed to be associated with end-organ deafness. Mygind produced perilymphatic hydrops associated with hearing changes of the end-organ type in experimental animals by creating excesses of water, salt, epinephrine and pilocarpine and by induced inflammation of the middle ear.

Hearing changes characteristic of end-organ deafness are: 1) Elevated threshold for bone conduction, 2) flat or rising (bass deafness) curve for air conduction threshold, 3) varying degrees of hearing loss, 4) poor discrimination for speech signals presented at high intensities, 5) recruitment of loudness for pure tones (best demonstrated by the binaural loudness balance test), 6) diplacusis binauralis dysharmonica and 7) decreased threshold for pain from acoustic stimuli.

The presence of end-organ deafness supports the diagnosis of Ménière's disease. The findings are variable and must be correlated with the clinical history. Hearing may be normal during remissions in the early stages of Ménière's disease and in cases without endolymphatic hydrops (pseudo-Ménière's disease). The findings of end-organ deafness are most prominent during the active phase of the disease; the findings become less marked in the late stages. This is presumably due to irreversible changes in the organ of Corti or the spiral ganglion. Evidence of end-organ deafness is absent in some cases of long-standing Ménière's disease.

In many instances deafness of sudden onset is of the end-organ type. Some patients who suddenly become deaf recover their hearing if the sudden deafness is of the end-organ type. They do not tend to recover hearing if the deafness is of nerve type.

Of the various signs of end-organ deafness those which gave the best correlation with the diagnosis of Ménière's disease were a flat curve for air conduction threshold, recruitment of loudness demonstrated by the binaural loudness balance test, poor discrimination and variability of hearing. Tests for diplacusis were difficult to interpret. Results of tests for a lowered threshold of pain correlated poorly with the clinical diagnosis.

DISCUSSION

DR. JOHN R. LINDSAY: Tests for recruitment and discrimination have become of wide interest in recent years; we are gradually beginning to understand them. It seems that in end-organ deafness the poor discrimination as shown by the P.B. maximum discrimination

scores goes along with the presence of recruitment; i.e., they have recruitment as shown by the loudness balance test and they have poor discrimination, and probably the two are related. We have been interested to find that when the lesion is beyond the end-organ, as in acoustic neuroma, and then probably also higher up in the cochlear pathways, there is a different situation. We have recently seen two cases, one an acoustic neuroma, the other an angle tumor in which there was a very good threshold of hearing for pure tones in the affected ear. We found no recruitment in those two cases. The first patient recognized that she could not understand speech over the telephone, yet her pure tone threshold level by air conduction was around the 8 or 10 decibel level; her threshold level for spondee words was around the same region, yet her discrimination for P.B. word lists was very poor. In that patient we used the Rush-Hughes recording and the discrimination score was low. Mr. Duncan Scott, who tested the case, found that by using another set of words spoken by a different voice the discrimination was relatively high. We find a large difference in the discrimination scores depending upon the recordings used. The second case showed similar findings; while there was a good threshold level for pure tones (i.e., up around 10 or 15 decibels), no recruitment and a good threshold level for spondee words, yet the discrimination score for the Rush-Hughes recordings of P.B. words was again away down.

It leads one to suspect that if we had a more difficult set of word lists with which to test these patients who have more centrally placed lesions, we might be able to demonstrate more clearly the loss of discrimination and this might then become a worth-while diagnostic sign. It has been recognized by some observers for a long time that in certain central lesions there might be relatively good hearing for pure tones but poor hearing for speech. Dr. Simonton showed one case of an acoustic neuroma with a fairly good threshold level for pure tones in which the discrimination score was 92 per cent. It makes one wonder whether, if he had had a more difficult list of works spoken by a less clear voice, it might not have been possible to detect poor discrimination. In a recent article by Bocca and associates concerning temporal lobe lesions, he reported that he was able to demonstrate poorer discrimination in the opposite ear, but it was only when the test was made difficult by the use of a filter that it would show up.

DR. SHERMAN SHAPIRO: The work which Dr. Simonton has done is very important. I think, however, from a practical standpoint what we need more than anything else is something to tell us at what level the lesion is present; in other words, we must be able

to diagnose a level above that of acoustic neuroma and that, of course, is what has been so difficult. I believe that Dr. Simonton will agree, speaking from a practical standpoint, that the average otologist finds little trouble in diagnosing the usual case of Ménière's disease. I would like to ask Dr. Simonton over how long a period he estimates the acute phase of end-organ deafness with the flat or the rising curve where there is no treatment.

DR. ROBERT HENNER: An interesting phenomenon to observe in testing an otosclerotic patient is that when you go from not hearing to hearing audiometrically, there will be a higher threshold loss of hearing; whereas, if you go from hearing to not hearing, lower thresholds will be obtained. This variability in threshold that exists in conductive hearing loss as compared to the sharpness of threshold in nerve deafness, is probably the same phenomenon that exhibits itself in the Luscher test. I am sure that people who are using this test will find it worth while. We are getting some valuable findings from it at present. Even better than the Luscher test for differentiating end-organ deafness, is the speech discrimination studies. Decreasing speech discrimination scores are the best indication of increased recruitment. I think it is part of the same phenomenon, but even more simple, and for that purpose the speechogram should be used more widely. If, for example, the speech discrimination scores become poorer as one raises the volume, one is dealing with marked recruitment.

Dr. Simonton mentioned the diagnosis of Ménière's disease without deafness in a few cases. I do not know how that diagnosis could be made. He also mentioned treatment. I have never known any therapy that I could say really works. Spontaneous remissions are the usual course, and this of course interferes with evaluation of the results of therapy. I have discarded all treatment, and feel that my ultimate results are no different than when drugs and salt free diets, etc., were used. I would like to know if anyone can definitely say that any treatment published to date has any value in the treatment of Ménière's disease.

DR. K. M. SIMONTON (closing): How long does a flattened or rising curve persist? This is an extremely variable thing, depending upon the severity of the manifestations of Ménière's disease, the frequency of the attacks, and perhaps some other factors. Some individuals maintain a variable hearing for many years; in others that variability fades out very quickly in the course of the disease. There are cases in whom deafness develops suddenly, who show evidence of end-organ type of deafness, who never recover the hearing. The

persistence of end-organ deafness is an extremely variable thing; I cannot give an answer in time, since it varies so much with different cases.

So far as the discrimination curve is concerned, it is not the percentage of discrimination that is significant in differential diagnosis of the type of hearing loss. Decrease in discrimination when intensity is increased suggests end-organ deafness. In nerve lesions the discrimination curve levels off when intensity is increased. I feel that there are many factors in discrimination; the poor discrimination is not a factor of an end-organ deafness alone. For instance in some observations we made on discrimination in quiet and noise, we had two men who had been railroad engineers, approximately the same age—65 and 70. They had practically identical pure tone curves both as to the shape of the curve and the threshold. One could discriminate quite well, the other extremely poorly, which undoubtedly was due to something other than the cochlea and the spiral ganglia. Perhaps the older individual with poorer discrimination had greater arteriosclerosis; perhaps his mental faculties as a whole were poorer. Each of these individuals maintained that he heard well in the noises made by a railroad engine; this was not confirmed by the laboratory data.

With reference to the question of the diagnosis of Ménière's disease without deafness, let us go back to Ménière's original papers. He described primary hearing loss in some individuals; primary vertigo in other individuals; and some in which the two manifestations appeared more or less simultaneously. I find many patients with Ménière's disease who have had two or three attacks of vertigo before developing loss of hearing, or who have developed loss of hearing and subsequently developed vertigo. The diagnosis of Ménière's disease without deafness is made by the presence of recurring vertigo of a whirling type, quite characteristic of that found in Ménière's disease, in the absence of evidence of cochlear change. When we make this diagnosis it probably indicates an individual who maintains vestibular symptoms without cochlear symptoms for a longer period than is usual.

I grant that the occurrence of spontaneous remissions makes evaluation of therapy difficult, yet one finds patterns of attacks and remissions in individual patients. One individual will have attacks at long intervals; another will have attacks at shorter intervals; or there will be a series of episodes and then a remission. Among those individuals who set up a pattern of attacks at short intervals, or a series of attacks followed by a remission, medical therapy often alters

the pattern of the disease by inducing early remission of symptoms. I, for one, am going to treat patients with Ménière's disease, and treat them very actively.

Meeting of Monday, January 3, 1955

THE PRESIDENT, DR. NORMAN LESHIN, IN THE CHAIR

Ossifying Fibroma of the Paranasal Sinuses

EMANUEL M. SKOLNIK, M.D.

AND

ELIO J. FORNATTO, M.D. (By Invitation)

CHICAGO, ILL.

(Complete paper appears on page 689)

DISCUSSION

DR. EMANUEL M. SKOLNIK: A few points might be stressed. On examination of the x-ray films the convexity of the lesion is more significant than the irregularity in each case. In the literature we find that most patients presenting this lesion are under the age of 30. Frozen sections are not of great value, and one learns to use clinical judgment rather than section. On examination at surgery, the tumor presents a very granular type of tumor which almost crunches when it is removed. The diplopia present was due to involvement of the intraorbital plate in two cases.

DR. BURTON J. SOBOROFF: We have seen two instances of such tumors, one in the frontal region and one in the antral region, both of which were very extensive. Each showed the same benign picture. One was in a relatively young man who had done a great deal of boxing in school and in the Navy; he developed a swelling in the antral region. The other was in a woman a little older. In each case we performed an extensive operation.

DR. ELMER A. FRIEDMAN: I should like to bring out a point mentioned by Dr. Fornatto about the relationship of these tumors to monostotic fibrodysplasia. About nine years ago a young boy was referred to me with a tumor occluding the right external canal. X-ray of the mastoid showed a destructive process of the right mastoid bone.

The tumor of the canal was biopsied, and the pathologic report was of ossifying fibroma. A mastoidectomy was done and the tumor, which involved the entire mastoid was removed as completely as possible. It had destroyed the posterior canal wall and occluded the canal. It was found to have enveloped the facial nerve. The facial nerve was laid bare from the horizontal canal to the stylomastoid foramen. It hung like a telephone wire without support. Postoperatively the patient had complete facial paralysis which cleared completely after several months. After great difficulty the pathologist rendered a diagnosis of ossifying fibroma, locally malignant. Because of this the patient was given postoperatively a cancerocidal dose of radium therapy. Healing was uneventful, the posterior canal wall reformed, the hearing became normal, and the patient is normal as of this time. Comparing the slides with subsequent reports in the literature I though it would have been better diagnosed as monostotic fibrous dysplasia. Had this diagnosis been made I would not have had so much anxiety, I would not have told the parents there was a malignant quality to the tumor, and I would not have used radium postoperatively.

DR. ELIO J. FORNATTO (closing): Dr. Soboroff mentioned a patient who had been a boxer. Very probably that lesion was monostotic fibrous dysplasia which represents an abnormal response of the bone to trauma.

As to total removal of the tumor—we did not remove it as widely as might have been done, and we did not get recurrence. The picture I showed was taken two years after operation and there was no sign of recurrence. We feel that we should not cause disfigurement by complete removal, but we do remove as much as possible without disfigurement. If it should recur, partial or total resection is indicated.

With regard to Dr. Friedman's case, there are two cases of ossifying fibroma of the mastoid reported in the literature. Unfortunately, we cannot make a clear-cut distinction between the two conditions. The only difference that exists is a prevalence of spherical islets in ossifying fibroma over the bony spicules characteristic of monostotic fibrous dysplasia. However, there are people who have demonstrated that there are intermittent stages between the trabecular and spherical types. Others have even considered the two terms as synonymous for the same condition. Nowadays, a histologic differentiation is impossible, in our opinion.

Otogenic Brain Abscess

MAURICE F. SNITMAN, M.D.
HARKISHEN SINGH, M.D.
JOHN H. GLADNEY, M.D. (By Invitation)
AND
JAMES R. SOFRANEC, M.D. (By Invitation)
CHICAGO, ILL.

(Abstract)

Two cases of otogenic brain abscess were presented with varying clinical pictures. The first patient was a 30 year old white male who developed an acute exacerbation of a chronic otitis media of the right ear. The chief symptoms were pain in the right temporal region associated with fever and chills. The presence of an increased spinal fluid pressure occasioned mastoid exploration where pus under pressure was found. The expected improvement following this surgery did not take place and, with the development of papilledema, a brain exploration was performed and a large temporal lobe abscess found and drained successfully.

The second patient was a 60 year old white male who developed an acute exacerbation of chronic otitis media of the left ear which did not respond to antibiotic measures. Accordingly, the mastoid was explored and here also pus under pressure was found in the mastoid cavity associated with a definite cholesteatome. The postoperative recovery was excellent, but fourteen days later he developed signs of intracranial pathology and brain exploration was advised. A large abscess of the left temporal lobe was uncovered and drainage was instituted, with a final excellent result. Recovery in both cases has been complete.

DISCUSSION

DR. ROBERT LEWY: I have a rather deep interest in both these cases since I was responsible for the initial radical mastoidectomy. Probably there are several questions that may be germane to this discussion. The most pertinent is, why were the patients not operated upon for brain abscess at an earlier date? There were not sufficient signs and symptoms to justify the diagnosis of brain abscess. The first case showed only somnolence, which was rather better after mas-

toidectomy. It was only when it became very suddenly more severe and papilledema was found, that we decided to put in a needle. Circumstances must justify such radical action. The next question is, when does one operate? Operation for brain abscess is performed when its presence is reasonably established. In the first case it was fairly clear that something was going on, and it was strongly suspected to be in the temporal lobe. The increase in somnolence, however, pointed to its being localized in the subtentorial area.

The second was more complicated in some respects, and not in others. One man thought the patient had lateral sinus thrombosis, which accounted for the septic curve. Others of us argued that the original septic curve was not vascular in origin. Then a second dissent caused us to call for a neurosurgeon. He did not feel that there was an abscess but that there was a vascular accident. That, combined with the temporary paralysis helped pretty well in localizing the lesion, but it was difficult in the face of an expert opinion to the contrary to insist that a brain abscess was present. The result, however, proved this hypothesis to be quite correct.

DR. THOMAS C. GALLOWAY: In the old days this would have been the opportunity for a long acrimonious debate about how to treat brain abscess. At Cook County Hospital, Pearlman said to operate on a brain abscess when you had a diagnosis. It was my feeling that operation should not be done until it was walled off. The danger of rupture into the ventricle and of disseminated meningitis made early intervention hazardous and I think it was an advantage to wait. But recently I saw Dr. Tarkington handle a brain abscess in a more modern way. He dissected out the abscess relatively easily and I think the danger of scar tissue and secondary infection is slight with antibiotics. Dr. Alexander of Vienna had been very proud of his early results half a century ago, but on following them up later found most of them in asylums. We can be very grateful for this better handling of otogenic disease.

DR. MAURICE F. SNITMAN (closing): I agree with Dr. Lewy about the question of delaying exploration because of the fact that in the first case all the symptoms, bradycardia, initial spinal fluid pressure of 300 mm of water, somnolence and low fever could well have been due to otitic hydrocephalus. I think most of us would have withheld surgery further. The second case was interesting in that the patient, having such a large abscess, following the mastoidectomy just went about his usual duties in a fairly normal manner, and the possibility of intracranial pathology would be difficult to suspect. In both cases one would expect some degree of facial weakness, but none

was found at any time. As a general rule, the fundamental therapy is drainage from the abscess through an adequate dural incision. We are very pleased with the postoperative progress of both patients.

Chondrosarcoma of the Nasal Cavity

BURTON J. SOBOROFF, M.D.

CHICAGO, ILL.

(Complete paper appears on page 718)

DISCUSSION

DR. LAWRENCE J. LAWSON: Dr. Soboroff has given us an excellent discussion of the character of this tumor. I encountered many of the same problems with a patient of mine, particularly with the inaccessibility of the tumor origin and the impossibility of wide margin removal.

My patient is now 80 years of age. She is very uncomfortable and requires continuous medication for pain. Dr. Gordon New removed the first tumor which originated from the left side of the nasal septum. The diagnosis was chondroma. She was not seen until she appeared five years later at our clinic. She complained of right nasal obstruction and pain over the right antrum. The septum had been invaded by the tumor, which had also extended toward and into the right antrum. It has since invaded the orbits and there is now complete nasal obstruction; she is blind from tumor pressure and tumor occlusion. A further report on this case will be given as opportunity permits. We have used deep x-ray therapy, which was for a time effective in decreasing the volume of the tumor. Perhaps extensive removal of the nasal septum at the time of the original operation might have eradicated the tumor.

DR. THOMAS C. GALLOWAY: I wonder if there may not be a better approach to this situation. Dr. Soper and I had a patient with a chondroma which had replaced the septum and sphenoid body. Operating within the capsule by a coring technique, I found everywhere smooth glistening capsule; there was no infiltration beyond it. That patient, who was in her seventies, went along for three or four years and died of something else.

I planned to operate on another case and was considering a technique which might have been satisfactory. It had a well defined peri-

chondrium of doubtful malignancy. I think some of these tumors must develop from the perichondrium and if one could approach them from the inside and use zinc chloride to destroy the perichondrial lining the result might be better. If the capsule is torn one gets dissemination. If one can destroy the tumor and capsule with diathermy I think it is advisable, but it is almost never possible.

DR. M. F. SNITMAN: The photomicrographs clearly indicate a diagnosis of chondrosarcoma, and the post mortem findings are evidences of its growth potentialities. However, even the diagnosis of a true chondroma should place the clinician on guard and institute radical measures. An exact determination of the sarcomatous qualities in a supposedly benign chondroma is very difficult, and the interpretation will vary among several pathologists. Our experiences with such tumors, although infrequent, have been very unpleasant.

DR. BURTON J. SOBOROFF (closing): It seems to be fairly well agreed in most of the literature, including that of Geschicter, Copeland, Coley and all the others, that the treatment of choice in chondrosarcoma is rather wide excision, in any case where they try to conserve function.

If we were fortunate enough to see these cases early, with the means at hand surgically, and the Fergusson type of incision which gives good exposure, I think wide surgical excision could be accomplished without extensive disfigurement and with much better results.

Meeting of Monday, February 7, 1955

THE PRESIDENT, DR. NORMAN LESHIN, IN THE CHAIR

Labyrinthine Surgery for Ménière's Disease

JOHN R. LINDSAY, M.D.

AND

KARL H. SIEDENTOP, M.D. (By Invitation)

CHICAGO, ILL.

(Abstract)

The experiences with different types of labyrinthine surgery for Ménière's disease at the University of Chicago for the years 1940 to 1954 were reviewed. The results of animal experiments and of

earlier operations on the labyrinth were reported before the American Academy of Ophthalmology and Otology in 1948. Although the animal experiments indicated that a differential destruction of the vestibular sense organs was feasible, the experience on humans was that any operation on the labyrinth which would consistently destroy vestibular function and thereby prevent the attacks of vertigo would destroy hearing in that ear as well. It was also recognized by Day that the relief of tinnitus, sometimes a distressing symptom in this diseases, was more likely to follow if cochlear function had been abolished. Both electrocoagulation (Day) and removal of membranous canal with its ampulla (Cawthorne) have been relatively successful operative procedures. In this series there were recurrences of vertigo in two out of nine cases after the former technique, and in one case out of ten after the latter technique. The hearing was destroyed in all cases. Tinnitus continued unabated in four of the former and in four of the latter group. In an effort to improve the results it was decided to adopt a more radical operative procedure on the labyrinth. This consisted of exenteration of the ampulla of the superior and horizontal semicircular canals thus making one large opening into the vestibule. The membranous structures in the vestibule were then ablated or destroyed. Coagulating current was not used. This procedure has been used in the last four cases with good results. In our experience the destructive operation on the labyrinth gives satisfactory results in selected cases of Ménière's disease and is a safe procedure. The proportion of cases in which the operation has been considered to be advisable has in recent years ranged between five and ten per cent.

DISCUSSION

DR. SHERMAN SHAPIRO: I have followed these operations with considerable interest for some years. I might say that I have not had any personal experience in labyrinthine surgery except for the Neumann operation for suppurative labyrinthitis, because I have never found a case where I felt justified in doing this. I have always suspected from my own limited experience that one could not do anything with the labyrinth without destroying the hearing, and I am happy that Dr. Lindsay brings this out so clearly, because I thing that will clear up a good deal of confusion. We must be prepared to tell such a patient that his hearing will be destroyed by operation.

A frequent problem involves the question of unilateral and bilateral Ménière's disease. Sometimes this is difficult to determine, and in many of these people one ear will be involved first and, owing to factors, systemic and otherwise, present in that particular individual, the other ear will become involved later. Practically, I find great

difficulty in even considering an operation of this type. For instance, one patient whom I saw today, I have had under observation for a number of years. He has practically no hearing in one ear; in the other ear he has enough hearing so that he can converse. He has shown many times a marked spontaneous and postural nystagmus, particularly with the Frenzel glasses. I do not know which ear is responsible for the Ménière's and I do not know whether this can be diagnosed. I do know, however, that this patient has been able to work and get along, even though he demonstrates some nystagmus most of the time. He is taking eight 50 mg nicotinic acid tablets daily, flushes beet red, but says the medicine does him good. I suppose the good ear will ultimately go deaf, but for some years at least he will be a functioning member of society.

I do not know of anyone who has been doing this operation in this vicinity except Dr. Lindsay, and I think he has done us a signal service in defining the strict limitations and the indications for it.

DR. ROBERT LEWY: We are much indebted to Dr. Lindsay for defining a better operation for intractable vertigo than we have had prior to this time. But there is a disparity which I am at a loss to explain. For eight years I have been working in a large public institution and we have not had any patients on whom the Cawthorne operation, or any modification of it, was done because of vertigo. I may be wrong, but I checked with another Michael Reese staff member and in the past ten years I find that no such operation has been performed there. In my private practice, which is not the most extensive, my percentage of patients with vertigo is fairly high, but the percentage who have required surgery is zero. I know that if any individual gets the reputation for doing a certain procedure, particularly if it is an esoteric procedure, cases tend to fall into his hands. Yet it seems to me there may be some variations of indications in various institutions. I am certain that there are patients who require this type of surgery, but in my experience I have seen none.

DR. MYRON M. HIPSKIND: I feel compelled, in view of the two previous discussants' remarks, to present the other side of the story. I wish to emphasize that Dr. Lindsay stated in the first part of his presentation that the surgical correction of vertigo was indicated only in those cases that fail to respond to medical therapy. Some of you may recall a motion picture presentation of a case of intractable labyrinthine hydrops which I presented before this Society last year. You may recall also that I pointed out the unfortunate loss of the fingers of the right hand which were amputated as a result of a fall during an attack of dizziness in which the patient, in an effort to break the fall, accidently plunged his hand into crushing mov-

ing gears. This man had a loss for serviceable hearing in the affected ear. He was totally incapacitated because of his recurring attacks of dizziness. Following the surgical procedure as outlined by Dr. Lindsay, the man regained his stability and has since been able to follow gainful employment. In those cases where there is a substantial danger to life, where medical management has failed to produce stabilization of balance, and where all serviceable hearing is lost, it is my feeling that they should be given an opportunity of surgical correction. I can speak of other patients who have had such a background in which surgery restored them to full functional and serviceable capacity.

DR. JOHN R. LINDSAY (closing): The operation I have described is a very simple modification of a labyrinthectomy. Regarding the number of cases seen by different otologists, I do not know all the factors contributing to this experience, but I am sure that each week I see two or three new cases of vertigo. I think this is a fair estimate without having gone over the cases accurately. The majority of these are not Ménière's disease, but are either postural vertigo or an attack of vertigo without auditory disturbance. I would estimate that we see twenty or more new patients a year that could be unmistakably diagnosed as Ménière's disease. It may be that they gravitate to certain doctors or clinics because of a special interest they are known to have in that problem.

Regarding the indications for surgery; one of the first pathologic specimens which came to me was from Dr. Boies of Minneapolis. It was from a man who fell and sustained an injury which resulted in my getting his bones. Another was a man who had not dared to walk out of his hotel alone for two or three years because of these attacks. Within four or five months after the operation he would go downtown or any place he wished. He made a rapid recovery for a man in his mid-seventies. There was another patient who on several occasions was literally thrown across the table into the lap of the person opposite. He called these attacks the "flip-flops" and is now very grateful because he has not done a flip-flop since. I just mention these because you might think that a person with Ménière's disease is not in danger. Another patient was driving along the road, had a sudden attack, and hit a telephone post. We agreed that something had to be done to prevent another such attack. I could tell you of more instances, but I only want you to realize that these people could not be controlled by other means and needed rehabilitation. However, I feel that the percentage of patients to whom this operation should be recommended is low, certainly less than ten per cent of the patients which we have seen in the past fifteen years.

GEORGE BACON WOOD

1871-1954

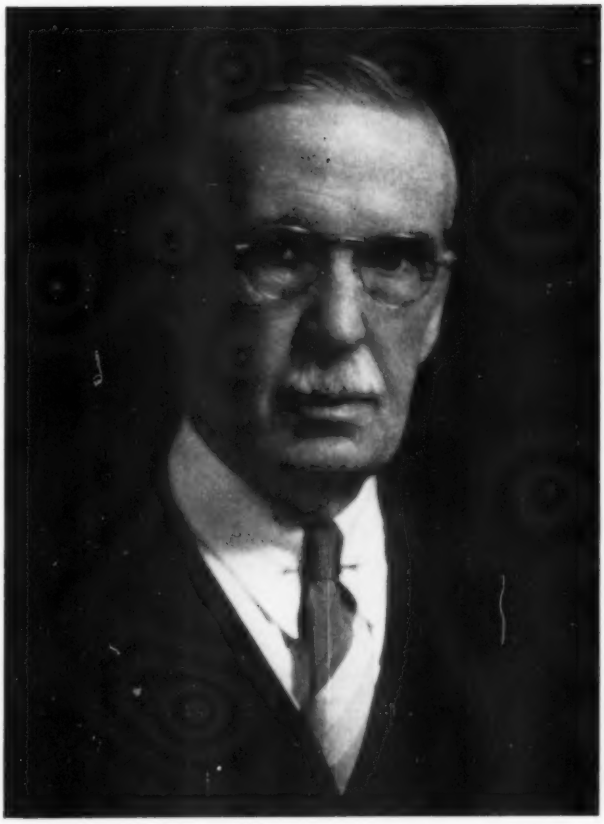
Dr. George B. Wood, known to many of his younger colleagues as Uncle George, was one of nature's noblemen. His father, Dr. Horatio C. Wood, was Professor of Materia Medica, and Clinical Professor of Nervous Diseases at the University of Pennsylvania. A number of other physicians of prominence were more distantly related. His mother, Eliza Huldah Longacre, was the daughter of James Boston Longacre and Eliza Stiles of Philadelphia. James Longacre was the Chief Engraver of the United States Mint and engraved the portrait of Benjamin Franklin for the first five cent stamp ever issued by our Government.

As a boy, Dr. Wood attended The William Penn Charter School and The George F. Martin School for Boys. In 1890 he was graduated from the Biological Department of the University of Pennsylvania and from the Medical School of the same Institution in 1894. Later he attended the University of Goettingen (1896) and the University of Berne (1897).

Before going abroad he interned for one and one-half years in the University Hospital in Philadelphia and on his return began the study of otolaryngology under the direction of Dr. Walter J. Freeman. During his professional life he was actively connected with the University, Graduate, Children's, Polyclinic, Howard, Orthopedic, St. Christopher's and St. Joseph's Hospitals and the Phipp's Institute.

In addition to this he was consultant to a number of other local schools and dispensaries. Later he was made Professor of Laryngology in the Graduate School of Medicine of the University of Pennsylvania. He was always a zealous teacher and loved dispensary work as a most valuable method of passing knowledge to the student. Even as Emeritus Professor in his late seventies and early eighties, once or twice a week he would put in an hour of two of dispensary teaching. The indigent to him were just as important as the wealthy.

He became a fellow of the American Laryngological Association in 1905 and served as President in 1938-39. He also belonged to The College of Physicians of Philadelphia, The American Medical Association, the Triological and the Philadelphia Pathological Societies. During World War I he served as major having charge of otolaryngology at Camp Mead for the duration.



GEORGE BACON WOOD

Dr. Wood was a delightful companion. His outside interests were many. He served as Trustee and President of the Arch Street Methodist Episcopal Church for many years. He was an outdoor man and loved nature in all its aspects. He was an expert fisherman, both salt and fresh water. He tied all his own trout and salmon flies with a perfection seldom obtained by a professional. He had a droll sense of humor and never hesitated to attempt to set one right if he felt you to be wrong, in a gentle manner however, that never could offend.

During his life he made more than eighty contributions to medical literature.

In 1906 he married Helen Foss of Philadelphia who survives him along with two sons, one daughter and six grandchildren.

K. M. H.

GORDON BALGARNIE NEW, M.D., D.D.S.

1885-1954

Dr. Gordon Balgarnie New died at the Methodist Hospital, Rochester, Minnesota, on October 28, 1954, after an illness of only a few days.

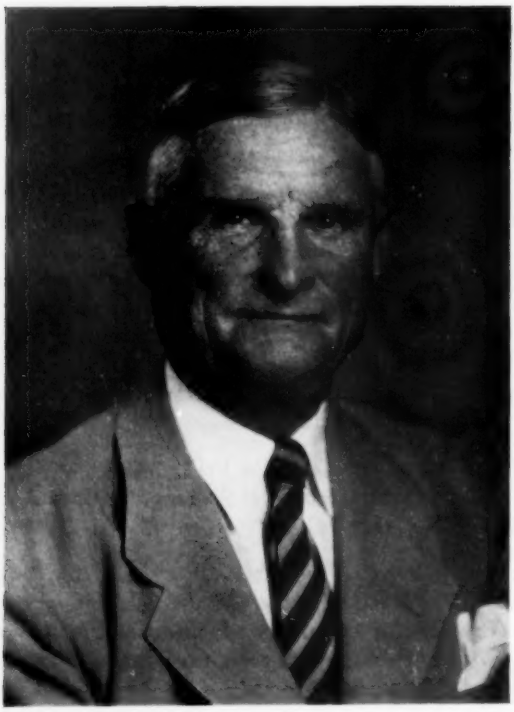
Dr. New was born in Hamilton, Ontario, Canada, on November 6, 1885. He attended the University of Toronto and received the degree of doctor of dental surgery in 1906, that of bachelor of medicine in 1909 and that of doctor of medicine in 1915. In 1909 and 1910 he was an intern in the City Hospital in Hamilton, Ontario.

He became affiliated with the Mayo Clinic in May, 1910, at which time he was appointed head of the section of postoperative treatment and an assistant in laryngology and rhinology. In February, 1911, he was appointed head of the Section of Laryngology, Oral and Plastic Surgery. When the Mayo Foundation was established as a part of the Graduate School of the University of Minnesota in 1915, he was appointed assistant professor of rhinology, laryngology and stomatology, and later professor of laryngology. In 1950 he became Professor Emeritus.

Dr. New was married to Miss Ethel Bailey on June 28, 1914. Mrs. New and two sons, Gordon, of Solvang, California, and Robert, of Lakewood, Ohio, survive.

He was considered an international authority on tumors of the nose and throat and was a pioneer in the fields of laryngology and plastic surgery in this country and abroad. He was extensively honored for his contributions to these branches of surgery. In 1949 he was given the Newcomb Award of the American Laryngological Association for his scientific contributions to otolaryngology. In 1952 he was awarded a silver plaque by the American Society of Plastic and Reconstructive Surgery for his contributions to plastic surgery; this award was the highest that could be given to a surgeon by that Society.

He was elected president of the American Association of Oral and Plastic Surgeons in 1939, president of the American Academy of Ophthalmology and Otolaryngology in 1945, president of the Pan-American Congress of Ophthalmology and Otolaryngology in 1946 and president of the American Laryngological Association in



GORDON BALGARNIE NEW

1950. In 1947 he served as second vice-president of the American College of Surgeons.

Dr. New was certified as a specialist in otolaryngology in 1925 by the American Board of Otolaryngology and as a specialist in plastic surgery in 1939 by the American Board of Plastic Surgery. In addition to fellowship in the American Laryngological Association, he was an active member of the American College of Surgeons, the American Medical Association, the American Laryngological, Rhinological and Otological Society, the American Academy of Ophthalmology and Otolaryngology, the American Society of Plastic and Reconstructive Surgery, the American Association of Plastic Surgeons, the Minnesota Academy of Ophthalmology and Otolaryngology, the Alumni Association of the Mayo Foundation and the Society of the Sigma Xi.

Those who were intimately acquainted with Gordon New will remember him professionally not only for his skill and dexterity as a surgeon, but also for his keen discernment in problems of clinical diagnosis. As an organizer and administrator, he was unexcelled. He was tremendously interested in all types of athletics and was an avid golf enthusiast.

In Dr. New's passing, surgeons in the fields of otolaryngology and plastic surgery alike have lost a sincere and earnest colleague who did much to advance the status of these specialties.

J. B. E.

Abstracts of Current Articles

EAR

The Rhesus Factor in Deafness.

Cavanagh, F.: Jour. of Laryngol. and Otol. 68:444 (July) 1954.

The designation "congenital" is not strictly correct when applied to deafness arising from Rh incompatability since the pathological process occurs during the first two weeks of life.

The Rhesus factor discovered by Landsteiner and Wiener in 1940 consists of a protein antigen on the surface of the red blood cells of 85 per cent of the white population. The factor was so named because the test is based on immune bodies in rabbit serum produced by injecting Macacus Rhesus (monkey) blood into rabbits. Fatal transfusion reactions in the presence of satisfactory type grouping can now be accounted for by Rh incompatability.

Erythroblastosis fetalis occurs when an Rh negative woman marries an Rh positive husband and bears Rh positive children. It is thought that some of the fetal erythrocytes pass through the placental barrier into the maternal blood and provoke antibodies. These antibodies in turn cross the placental barrier and enter the fetal blood. The development of antibodies, although it varies greatly, is rarely a factor in the first and seldom in the second pregnancy. The author notes personal exceptions to this accepted rule however.

Erythroblastosis fetalis includes:

a. Hydrops fetalis. Infant usually dies in utero or at birth.

b. Icterus gravis. Severe juandice soon after birth frequently associated with neurological complications.

c. Congenital Hemolytic Anemia. Hemoglobin falls rapidly after birth and repeated transfusions are needed.

Pathologically the principal problem is anemia and jaundice appearing a few hours after birth. Autopsies show a characteristic pigmentation of the central nervous system in those infants dying a few days after birth. The region of pigmentation involves cortical as well as basal centers including eighth nerve nuclei.

Infants dying at birth do not show this pigmentation. Those surviving longer periods show degeneration and scar tissue in the pigmented regions.

Clinically the infant may be normal at birth but in a matter of hours develops bruising, jaundic pallor, hepatomegaly and splenomegaly.

If allowed to continue neurological signs may include: Drowsiness, failure to feed, opisthotonos, hypotonia, unusual eye movements, convulsions, athetosis, apnea and cyanosis, cerebral cry and spasticity. This clinically constitutes kernicterus or the neurological complication of Rh incompatability and hearing loss of the nerve type is not an uncommon complication of this condition. The severity of the neurological symptoms is in no way indicative of the degree of eventual disability.

The treatment consists of exchange transfusions 10 cc at a time over a two hour period beginning about three hours after birth.

The author analyses a series of 114 "Rhesus" babies of which six (5 per cent) were deaf, and a second series of 118 deaf children in which three cases of Rh sensitization were found.

N.B.: "Deaf" as used in this paper implies severe hearing difficulty.

SOOY.

Long-Term Results with the Fenestration Operation.

Campbell, Edward H.: A. M. A. Arch. Otolaryng. 60:265-276 (Sept.) 1954.

The author points out that today evidence of proof of the substantial results obtainable by the fenestration operation is not necessary, whereas ten or twelve years ago there was considerable skepticism on the part of some physicians and even otolaryngologists as to its value. The technical details have changed some over these years but the basic principles have remained the same. Today's high degree of success is dependent not only on a highly trained surgeon's operative technique but upon the judgment exercised in the selection of patients suitable for operation.

Campbell presents a statistical study to indicate little or no loss of hearing from the first to the sixth or seventh postoperative year in successful cases and offers explanations of further hearing loss in certain other fenestration cases. He discusses studies published by

Shambaugh and House and presents his own series of 200 fenestration operations studied over a period of five to eight years subsequent to operation with frequent audiograms.

The 200 fenestration patients have been grouped in three categories:

1. The group of 130 patients in which the operation had been successful (improvement to practical hearing level for the three speech frequencies). In this group the average decibel loss preoperatively was 49.2 for the three speech frequencies; the average decibel loss one year postoperatively was 27.1 db, two years postoperatively was 28.9 db and five years postoperatively was 27.6 db. Of 43 who had audiograms seven years after operation, the average preoperative decibel loss was 48.6, the average loss one year after operation was 24.5 db, five years postoperatively was 26.5 db and seven or more years after operation was 28.2 db.

2. The group of 49 patients whose operations were not considered successful (hearing either not improved to practical hearing level or satisfactory improvement followed by partial loss of improvement). Here the average decibel loss before operation was 50.6, the average loss one year postoperatively was 42.8 db, two years postoperatively was 46.4 and five years 49.2 db.

3. The group of 21 patients who attained improvement to a practical level of hearing, but experienced subsequent loss of hearing to the preoperative level or below. All in this group had the fenestrum revised. The average preoperative loss in this group was 45.8 db, the average loss just before revision was 50 db. Six of the 21 revisions were successful and of the remaining 15 cases in which the first revision was not successful, six had second revisions, of which three were successful; 45 per cent of this group of 21 eventually were classified as successful.

The author concluded that this study of hearing results over a period of five or more years following fenestration operation indicated that in the successful case hearing improvement present a year after operation can be maintained at approximately that level for a period of five or more years unless other factors not connected with the original condition or operation intervene to impair the hearing. In the unsuccessful cases, a further loss of hearing during the years subsequent to operation can be anticipated.

Some factors involved in evaluating postoperative hearing results are discussed at considerable length and these factors include the psychological reactions of the patient, temporary inflammatory or

allergic conditions of the nose and ears, secretory otitis, the degree of tinnitus present, emotional and nervous disorders, labyrinthine hypertension, auditory nerve degeneration and further advancement of the otosclerotic process. Some of the causes of failure are also dealt with in considerable detail. Closure of the fenestra subsequent to operation the author feels is probably the commonest cause of failure with postoperative serous labyrinthitis or labyrinthosis second in order of frequency. Some factors are dependent upon the skill of the surgeon, others are not. A wrong preoperative diagnosis, leading to operation upon an ear showing perceptive hearing loss may occur and the reasons for this are outlined. Primary or secondary infections of the ear cavity may be a major cause of failure. The presence of a perforation in the tympanic membrane may be another reason for failure but with healing the hearing improves to the anticipated level.

HILDING.

Injuries of the Eighth Nerve in the Evolution of the Influenza.

Saxa, Vojtech: Ceskoslov. Otolaryng. 2:4:226, 1953.

The virus of influenza penetrates the organism through the epithelial cells of the upper respiratory tract and has frequently a special affinity to the central nervous system (encephalitis and its sequellae).

The cochlear nerve is injured much more frequently than the vestibular; the explanation of this fact is attributed to the very tiny structure of its branches, as compared to the vestibular.

The consensus is that the nerve is injured by the toxic action of the virus and interference with the blood supply.

In some patients there exists a discrepancy between hearing and the findings of the audiogram. Speech reception is poor while pure tones are heard very well, suggesting involvement of the cortical "analyser." This can progress to deafness.

Associated lesions of fifth, seventh and occipital nerves explain the auricular pains in many cases.

The anamnesis is of high importance to the later diagnosis.

The author ends by describing three cases: two unilateral and one bilateral. In one case there existed a diminished vestibular sensitivity.

The author tried treatment with dehydration, high doses of pilocarpine for increase of the secretions, 40 per cent glucose, B-1 histamine, without success.

SOBIESKI.

Acute Otitis Media—Comparative Therapeutic Results of Sulphonamide and Penicillin Administered in Vaccine Forms.

Rudberg, R. D.: Acta Oto-laryng., Suppl. 113.

As the result of a study of 1365 cases of acute uncomplicated otitis media, the author concludes that penicillin by the oral route is as effective as when administered parenterally. Combined therapy of penicillin and sulphonamide is as effective, but no more so, than penicillin alone. Poorer results follow simple sulphonamide therapy. Conservative treatment was the least effective.

HILL.

Cholinesterase Inhibitors and Vestibular Function—A Study of a Vestibular Syndrome in Guinea-pigs Caused by Intracarotid Centripetal Injection of Cholinesterose Inhibitors and Cholinesters.

Diament, H.: Acta Oto-laryng., Suppl. 111.

Employing the injection technique of Skoog the author studied the vestibular syndrome produced by the injection of various cholinesters and cholinesterase inhibitors as well as the effect on the syndrome of injected antidotes against cholinesterase inhibitors. Histological examinations and the determinations of cholinesterase activity in different parts of the brain were carried out.

This is a most exhaustive study which suggests that the chemical transmission in the central vestibular sytem is cholinergic. Cholinesters other than acetylcholine can produce vestibular syndromes. The syndrome results from excitation of the effector cells of the injected side. Paralysis of these cells also produces a syndrome directed to the opposite side, originating from the intact effect on cells of that side. It also suggests that the syndrome originates in the region of the vestibular nuclei. At least this region must be intact for the syndrome to be produced.

HILL.

On the Chemistry of the Endolymph.

Vilstrup, T. E. G. and Jensen, C. E.: Acta Oto-laryng., Suppl. 109, pp. 200-201.

From experiments on the endolymph of sharks in which the viscosity changed upon incubation with hyaluronidase it was felt that the viscosity of the endolymph is due to hyaluronic acid.

HILL.

Modified Portmann Operation on the Endolymphatic Sac for Treatment of Mèniére's Disease.

Yamakawa, K. and Naito, T.: The Jour. of Otolaryng. (Tokyo) 26:7 (Jan.) 1954. 1954.

Portmann's operation on the saccus endolymphaticus has closed the fenestra in some instances and failed its objective. To overcome this complication, Yamakawa and Naito recorded their modification on this procedure.

The operation is performed by a post-auricular approach as in a classical Schwartz mastoidectomy. When the saccus is indentified, an inverted "L" shaped incision is made in the dura over the cerebellum next to the sac. This dural flap is lifted so as to gain an access to the sac on the cerebellar side. When a window is cut out from the wall of the saccus there is a free flow of the endolymph, but usually within five minutes, it ceases to run. The incised flap of the dura is then replaced and sutured.

According to these authors, they have operated on 12 cases of Ménière's disease, eight of whom obtained complete relief from tinnitus, vertigo without sacrificing the sense of hearing, and four were improved. The operative technique is shown by several drawings.

HARA.

NOSE

Ethmoidectomy and Allergic Rhinitis.

Riccabona, A.: Zeitschr. f. Ohrenheilk. Vienna 88:201 (July-Sept.) 1954.

An approach to the problem of the treatment of resistant allergic rhinitis is presented. Numerous patients whose typical nasal allergy resists various antihistamines and desensitization attempts are seen in every otolaryngologist's office. The theoretical possibility of an al-

lergy to bacteria somewhere in the upper respiratory tract in a large number of such patients is brought to mind.

The search for a focal infection of the paranasal sinuses by roentgen films may often show negative results. The author reminds the reader, however, that some ethmoid infections are radiologically detectable only with great difficulty, if at all.

The brief case histories of nine patients with allergic rhinitis are presented. All these patients gave a past history of purulent discharge from the nose, or proven sinus infection. The infections in these cases occurred years ago, cleared up clinically, and gave way to chronic watery discharge and typical nasal allergy. The author advised ethmoidectomy in the presented nine cases in the absence of roentgenological findings. Bilateral operation was done in most cases. Patients with obvious unilateral complaints or clear past history of unilateral infection underwent unilateral ethmoidectomy.

The histological and bacteriological examination of the ethmoid linings revealed in all cases infection with various organisms, as streptococci, pneumococci, coli bacteria.

All presented cases were free of allergic symptoms following the operation. Vasoconstrictor medication like Privine® had caused poor circulation in the ethmoid mucosa, the author explains, especially since vasoconstrictors so frequently are used over a period of many months. Due to this poor circulation the natural body defenses as well as the previously administered antibiotics were inadequate. A focus of infection remained, and the bacteria became the allergen for a chronic allergic rhinitis.

FISCHER.

THROAT

Cinefluorographic Analysis of the Mechanism of Swallowing.

Ramsey, G. H., Watson, J. S., and Gramiak, R., and Weinberg, S. A.: Radiol. 64:4: 498-518 (Apr.) 1955.

The authors made cinefluorographic studies of the swallowing act in 300 individuals. One-half were considered normal, varied in age from a few weeks to eighty years, and were of both sexes. Various positions and projections were used. Contrast medium was a mixture of barium sulfate and water. Examinations were recorded on 35 mm film at 30 to 60 frames per second. Thirty-seven radiographs depict the passage of the bolus into the oral pharynx, stripping

wave of the oral pharynx, closure of the nasal pharynx, entrance of the bolus into the oral pharynx, propelling of the bolus through the pharynx, return of the pharynx to the resting stage, elevation and closure of the larynx and descent of the bolus down the esophagus. The authors note that with the exception of a few constant reflex relationships the swallowing events vary considerably in different conditions. They emphasize that this must be taken into account in distinguishing between the unusual and the abnormal. The anatomical physiology of the act of deglutition is reviewed. Twenty-two references are cited.

JORSTAD.

Giant Tonsillar Calculus.

Soraluce, J.: Bol. Espan. Otoorrinolaringol. Broncoesofagol. 7:37-44, 1954.

Tonsillar calculus is a rare complication of chronic tonsillitis. If the calculus is large it produces the sensation of a foreign body, hoarseness and acute pain on swallowing.

In the case of an eleven-year-old girl, calculus of a tonsillar crypt was accompanied by sore throat, pus and adenitis. After ejection of the calculus during an attack of nausea the condition improved. The crypt was filled with caseous matter. Six months later pressure still elicited yellow pus and the lymph glands were enlarged but not painful on pressure. The calculus was hard, white, rough, granular and cone-shaped. It weighed 12.04 gm and measured 3.2 by 2.5 cm. On analysis it proved to be calcium carbonate.

The second patient, a woman seventy-five years old, had previously had recurrent renal lithiasis. The most recent attack, two years previously, had not resulted in expulsion of the stone which was found by roentgenographic examination to occupy the entire right kidney pelvis. A month previous to examination she had developed pharyngeal soreness, pain on deglutition, radiating into the ears, eyes and molars. A yellowish-white hard mass as large as an olive pit, weighing 0.16 gm covered with malodorous yellow pus, was extracted from the left tonsillar crypt with forceps. Caseous material was later removed from the crater left by the calculus. The stone consisted of calcium phosphate with organic matter. The renal calculus had the same composition but without organic material.

These calculi are sometimes formed by dehydration of the caseous tonsillar exudate, which forms a nucleus for the deposit of calcium

salts. As a rule they are small weighing less than 1 gm although occasionally they are as large as 5 to 8 gm, or even in one case, 18 gm. They usually accompany chronic tonsillitis and are not associated with calculi of other organs except in cases of calculous diathesis. Tonsillar abscess is a frequent complication.

HIGBEE.

Fusion of the Vocal Cords After Protracted Endotracheal Intubation.

Zwergius, E.: Acta Oto-laryng., Suppl. 109, pp. 210-214.

Two cases of acute barbiturate poisoning underwent endotracheal intubation. One was intubated for 146 hours with 8 changes of the tube. The second was intubated 258 hours with 14 changes. Both cases developed obstruction and required tracheotomy. Fusion of the vocal cords were found later, in the anterior third in one, and in the middle third in the other. In the latter case the fusion was severed. In both cases there was limited abduction, hoarseness and dyspnea on exertion.

Early tracheotomy would have been preferable to any such prolonged period of intubation.

HILL.

LARYNX

Demonstration in Man of the Existence in the Recurrent Nerve of Potentials of Motor Action Synchronous with the Vibration of the Vocal Cords.

Moulonguet, A., Laget, P. and Husson, R.: Ann. de l'Oto Laryngol. 71:5-6:365 (May) 1954.

The authors review the literature and explain briefly the experimental research of Lermoyez, the discovery of stroboscopy by Oertel, the myo-elastic theory of Ewald. Lindemann, in 1930, observed electrical potentials in the laryngeal nerves of the dog and deduced the existence of currents of stimuli, which would be afferent in the superior and efferent in the inferior laryngeal nerve. Later Heymann, in Frankfort and Dumont, in Paris, recorded the active vibrations of vocal cords, and how they could be provoked by short stimulations of the nerve.

Frequent electric stimulations (10-15 per second) begin to provoke the fusion of the movements and 50 stimulations per second induce a complete tetanization of the vocal cords.

In 1950, Husson proposed the theory that the vibrations of vocal cords are directly controlled by encephalic stimuli, and muscular fibers of vocal cords respond on each stimulus transmitted through the recurrent nerve.

This hypothesis was verified: In the dog by P. Laget, and in mankind by A. Moulonguet.

P. Laget connected the isolated recurrent nerve with a special electrode, and using a stimuli of one millisecond in the discharges of one-third of a second, the vocal cords were examined with a stroboscope. It was stated that with the stimulus the vocal cord approaches toward the midline, vibrating during the discharge at the frequency of the stimuli. The amplitude of vibration was one-half millimeter with a frequency of stimuli of 100 per second, much smaller with 400 per second, and diminished with the further increase of frequency. The maximal frequency to which the vocal cord was able to respond by distinguishable movements was between 400 and 500 per second.

In higher frequencies of stimuli the vocal cord responds only after the end of its own refractory period. In a very weak stimulation, there exists a "bi-phasic" response, and the frequency of vibration can reach 900 to 1000 per sec. This "bi-phasic" response is identical to that described by Stevens and Davis for the cochlear nerve. Each axone commands a very small group of muscular fibrils of the internal thyroarytenoid muscle, therefore, a "bi-phasic" stimulation can produce contraction of different muscular fibrils in two phases.

Similarly the possibility of "tri-phasic" function is postulated where each stimulus provokes the contraction of one-third of the muscular fibers only.

Using a specially constructed electrical and other instrumentation, the authors were able to undertake an "experimentl examination," of a patient undergoing laryngectomy for cancer with a relatively well preserved voice.

Under local anesthesia a careful dissection of the left recurrent laryngeal nerve was performed. The nerve was connected to two electrodes. The patient was asked to pronounce the sound "A" and the oscillographic movements were photographed.

A homorythmicity (the same rhythm) for the voice and the electric action potentials were recorded during all the tests. This action potential was seen on the photographic films some hundredth of a second before the appearance of the voice. The authors consider it to be a proof of reality of the motor potential and not of "an electrophonic effect." (The film of vocal vibrations is published in the text.)

The axones of mammals have a refractory phase of two milliseconds; therefore, the maximum vibration of vocal cords cannot exceed 500 per second. Above the "Do-4" tenors are obliged to use "bi-phasic" forms of vibrations, giving a "falsetto" voice. At this stage the x-rays and stroboscopy show a brisk decontracture of all the muscular apparatus, the intensity of cortical stimulation diminishes becoming "bi-phasic."

SOBIESKI.

Scheer's Operation in the Treatment of Paralysis of the Laryngeal Abductors.

Bol. Espan. Otorrinolaringol. Broncoesofagol. 7:45-46, 1954.

The various techniques commonly used to restore permeability of the respiratory passages and to correct phonation are rendered difficult by the dimensions of the larynx, and the postoperative respiratory disturbances which may ensue. Scheer's method attempts to overcome the disadvantages by gaining access to the larynx by means of thyrotomy. Following regional local anesthesia, tracheotomy is performed as low as possible, followed by classical thyrotomy along the midline. The exposed larynx is transfixed with a 2 mm steel thread anterior to the vocal process of the arytenoid. An incision is then made along the anterior border of the sternocleidomastoid, behind the thyroid wing, exposing the posterior border of the cartilage, the periosteum of which is reduced with a fine curette. A Reverdin needle is passed through the incision across the cartilage, drawing the end of it slightly upward behind the corresponding cord. A steel thread is then inserted and is temporarily left in the eye of the needle to ensure good traction and for subsequent tying.

Section of the vocal process is then carried out with fine scissors at the level of the arytenoids which are extirpated in toto. Subperichondral dissection of the cricoarytenoid articulation permits its displacement facilitating the extirpation of the arytenoids. The operative wound is closed with a single suture, with a No. 00 catgut.

The same technique can be employed in both unilateral and bilateral involvement. Postoperative edema of the larynx disappears within two weeks after intervention. The condition of the larynx should be observed by laryngoscopy at intervals.

One advantage of this method is that the steel thread can be readily drawn taut soon after operation through the skin incision. This aids respiratory function and phonation. Results obtained by Scheer and by others have been favorable.

HIGBEE.

ESOPHAGUS

Roentgenologic Study of the Lower Esophagus and Esophagogastric Junction.

Poppel, M. H., Zaino, C., and Lentino, Walter: Radiol. 64:5:690-700 (May) 1955.

The authors describe an olive oil barium contrast technique as a great aid in demonstrating the mucosal pattern of the lower esophagus as visualized by radiography. Thirteen radiographs and five line drawings demonstrate findings in the normal, in spasm of the lower esophagus, hiatus hernia, and ulcer in a case of scleroderma. The findings in these conditions are discussed. Five hundred unselected cases were studied. Positive findings were obtained in 67, and 54 of these in whom a complete follow-up was obtained 41 (81 per cent) had abnormalities overlooked in the conventional examination. Twelve references are cited.

JORSTAD.

MISCELLANEOUS

Roentgen Characteristics of Osteogenic Sarcoma of the Jaw.

Sherman, Robert S., and Melamed, Myron: Radiol. 64:4:519-527 (Apr.) 1955.

The authors report 19 "typical" osteogenic sarcomas of the jaw in which clinical features, x-ray diagnoses and histologic interpretations offered satisfactory correlation, and seven additional tumors which present one or more unusual aspects. In the interpretation of roentgen findings the authors evolved a broad grouping of three principal types: Sclerosing, lytic, and mixed. The correlation of radiographic and histologic findings is discussed. Case reports of the seven tumors were added to indicate the difficulty sometimes

encountered in establishing a final diagnosis. Twelve figures illustrate the roentgen and histologic findings. Twelve references are cited.

JORSTAD.

Plasma Cell Tumors of the Upper Respiratory Tract.

Boyes Korkis, F.: Jour. Laryngol. and Otol. 68:517 (Aug.) 1954.

The rarity of plasma cell tumors arising in the upper respiratory tract is stressed. A case is reported with studies over a four year period involving the ad seriatum appearance of lesions in the laryngeal ventricle, trachea and uvula.

The literature and histopathology are briefly reviewed.

SOOY.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

The Sixth International Congress of Otolaryngology will be held in Washington, D. C., at the Hotel Statler from Sunday, May 5, through Friday, May 10, 1957. Representatives of the five national societies of otolaryngology in the United States elected Arthur W. Proetz, M.D., St. Louis, President, and Paul H. Holinger, M.D., Chicago, General Secretary of the Congress. Other officers and members of the Organizing Committee are as follows:

Vice-Presidents (each from one of the five sponsoring national societies, as liaison representative)

Louis H. Clerf, M.D. St. Petersburg, Fla.	American Broncho-Esophagological Association
Dean M. Lierle, M.D. Iowa City, Iowa	American Academy of Ophthalmology and Otolaryngology
John R. Lindsay, M.D. Chicago, Ill.	American Otological Society
C. Stewart Nash, M.D. Rochester, N. Y.	American Laryngological, Rhinological and Otological Society, Inc.
Harry P. Schenck, M.D. Philadelphia, Pa.	American Laryngological Association

Treasurer	Frederick T. Hill, M. D., Waterville, Maine
Secretary for Local Arrangements	Victor R. Alfaro, M.D., Washington, D.C.
Secretary for Program	Lawrence R. Boies, M.D., Minneapolis, Minn.
Secretary for Public Relations	A. C. Furstenberg, M.D., Ann Arbor, Mich.
Secretary for Publicity	Howard P. House, M.D., Los Angeles, Calif.
Editor, International Directory of Otolaryngology	Chevalier L. Jackson, M.D., Philadelphia, Pa.
Secretary for Publications	Bernard J. McMahon, M.D., St. Louis, Mo.

Secretary for Social Affairs	Lewis F. Morrison, M.D., San Francisco, Calif.
Secretary for Finance	LeRoy A. Schall, M.D., Boston, Mass.
Secretary for Foreign Relations	George E. Shambaugh, Jr., M.D., Chicago, Ill.

Committees for other special purposes are being appointed as these are required.

The International Congresses are held every four years and each Congress is a separate entity. The host country is entirely responsible for the organization, development and financing of the Congress. Every participating country may send two official delegates elected by its national society of otolaryngology. These constitute the International Committee which considers invitations from various countries for the following Congress and official business other than that of the organization and management of any specific congress.

The International Committee at its meeting in Amsterdam, June 10, 1953, appointed an Interim Committee to function between the Fifth and Sixth Congresses. This committee consists of the President and Secretary of the Fifth Congress, Prof. Eelco Huizinga, Groningen, Netherlands, and Mr. W. H. Struben, Amsterdam, and the President and Secretary of the Sixth Congress, Dr. Arthur W. Proetz and Dr. Paul H. Holinger.

PREVIOUS CONGRESSES

	Date	Place	President	Countries	Members	Papers	Films
I.	1928	Copenhagen	Schmiegelow	41	600	—	—
II.	1932	Madrid	Tapia	48	—	—	—
III.	1936	Berlin	von Eicken	—	—	148	—
IV.	1949	London	Negus	45	1304	156	25
V.	1953	Amsterdam	Huizinga	39	700	160	39

THE SIXTH CONGRESS

The Sixth Congress at which all American otolaryngologists will be hosts will open on Sunday, May 5, 1957, with an Inaugural Ceremony at Constitution Hall followed by a reception at the Pan American Union. The scientific program will get under way on Monday, May 6. It will consist of three Plenary Sessions, on Monday, Wednes-

day and Friday mornings, which will be devoted to the discussion of selected subjects. While these are in progress no other scientific session will be held. The subjects which have been selected for these sessions are:

I. Chronic Suppuration of the Temporal Bone

II. Collagen Disorders of the Respiratory Tract

III. Papilloma of the Larynx

Outstanding, internationally recognized authorities will open each of these discussions.

On each afternoon and on Tuesday and Thursday mornings there will be five simultaneous sessions at which voluntary papers will be presented.

A program of medical films will be presented each morning and afternoon except during the Plenary Sessions. There will be scientific and technical exhibits.

The official languages of the Congress are English, French, German, Spanish.

A full program of social events is planned, and post-Congress tours to certain American medical centers are under consideration.

Invitations in the four official languages have been sent to the otolaryngologists of record throughout the world and a large attendance is expected.

Communications should be addressed to the office of the General Secretary, Paul H. Holinger, M.D., 700 North Michigan Avenue, Chicago 11, Illinois, U.S.A.

Notices

AMERICAN LARYNGOLOGICAL ASSOCIATION

CASSELBERRY PRIZE

A sufficient sum having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available, in part or as a whole, for a prize award. Theses must be in the hands of the Secretary, Dr. Harry P. Schenck, 326 South 19th Street, Philadelphia, Pa., before March 1, 1956.

Copies of the Transactions of the American Laryngological Association are available for general distribution at $8.00 a copy. Please send request with check to:

Dr. Edwin N. Broyles,
Editor Transactions
1100 North Charles St.
Baltimore 1, Md.

ACADEMY HOME STUDY COURSES

The 1955-1956 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, began on September 1 and will continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota. Registrations should be completed before August 15.

TULANE UNIVERSITY OF LOUISIANA
SCHOOL OF MEDICINE

The three year residency in otolaryngology offered at Charity Hospital of Louisiana at New Orleans on the Tulane University of Louisiana School of Medicine service is designed to qualify the holder for the examinations of the American Board of Otolaryngology and the practice of all phases of otolaryngology and endoscopy.

Candidates must be graduates of a class A medical school and must have completed a minimum of one year of general internship. An additional year of residency in internal medicine or general surgery is desirable but not essential.

All work is under the direct supervision of members of the Tulane Department of Otolaryngology, who are also members of the Charity Hospital Otolaryngological Staff; they are available at all times for instruction and guidance. Basic sciences are offered throughout each year of the residency during the academic year. The resident also participates in the program of the Speech and Hearing Center at the Tulane University School of Medicine.

The hospital year extends from July 1 of one year to June 30 of the following year.

Applications should be addressed to the Chairman of the Department of Otolaryngology, Tulane University of Louisiana School of Medicine, 1430 Tulane Ave., New Orleans 12, Louisiana.

UNIVERSITY OF ILLINOIS

The next Bronchoesophagology Course to be given by the University of Illinois College of Medicine is scheduled for the period October 24 to November 5, 1955. The course is under the direction of Dr. Paul H. Holinger.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

TEMPLE UNIVERSITY

Temple University offers a postgraduate course in broncho-esophagology October 17-28, 1955 and a postgraduate course in laryngology and laryngeal surgery September 19-30, 1955.

These courses are to be given in the Department of Laryngology and Broncho-esophagology, Temple University Hospital and School of Medicine, under the direction of Doctors Chevalier L. Jackson and Charles M. Norris. The tuition fee for each course is $250.00. For further information please apply to Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.

MEDING AWARD

On the evening of October 28, 1955, The Harlem Eye and Ear Hospital will bestow upon Dr. Franz Altmann, the Dr. Charles Bramman Meding Award. This award is granted annually to a person judged to have made an important contribution to the advancement of Otolaryngology or Ophthalmology.

Dr. Altmann's film, "Embryology of the Ear," was considered an outstanding achievement. It will be shown at the hospital auditorium at the time of the presentation.

OFFICERS

OF THE

NATIONAL OTOLARYNGOLOGICAL SOCIETIES

American Academy of Ophthalmology and Otolaryngology

President: Dr. Algernon B. Reese, New York.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.

American Board of Otolaryngology

President: Dr. Frederick T. Hill, Professional Bldg., Waterville, Maine.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Iowa.
Next examination: Palmer House, Chicago, October 3-7, 1955.

American Broncho-Esophagological Association

President: Dr. Daniel S. Cunning, 115 E. 65th St., New York 21, N. Y.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3, Pa.
Meeting: Mount Royal Hotel, Montreal, May 15-16, 1956.

American Laryngological Association

President: Dr. Bernard J. McMahon, St. Louis.
Secretary: Dr. Harry P. Schenck, 326 South 19th St., Philadelphia, Pa.
Meeting: Seigniory Club, Montreal, May, 1956.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Dean M. Lierle, Iowa City.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Meeting: Mount Royal Hotel, Montreal, May, 1956.

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. R. Lindsay, Chicago, Ill.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.

American Otological Society

President: William J. McNally, M.D., 1509 Sherbrooke Street West, Montreal 25, Quebec, Canada.
Secretary-Treasurer: Lawrence R. Boies, M.D., University of Minnesota Hospitals, Minneapolis 14, Minnesota.
Meeting: Seigniory Club, Montreal, Quebec, Canada, May 11-12, 1956.

The American Society of Ophthalmologic and Otolaryngologic Allergy

President: F. Lambert McGannon, Lakewood, O.
Secretary-Treasurer: Michael H. Barone, M.D., Buffalo, N. Y.
Meeting: Palmer House, Chicago, Ill., October, 1955.

VI International Congress of Otolaryngology

President: Arthur W. Proetz, M.D., St. Louis, U. S. A.
Secretary-General: Paul H. Holinger, M.D., 700 North Michigan Ave., Chicago 11, Ill., U. S. A.
Meeting: Washington, D. C., May 5-10, 1957.

International Bronchoesophagological Society

President: Dr. Antonio da Costa Quinta, Lisbon, Portugal.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
Meeting: IV International Congress, Buenos Aires, October 28-29, 1955.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. Juan M. Tato, Buenos Aires.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa.
President of the Congress: Dr. J. H. Font, Puerto Rico.
Secretary of the Congress: Dr. C. E. Muñoz MacCormick, Santurce 29, Puerto Rico.
Meeting: San Juan, April 8-12, 1956.

Pacific Coast Oto-Ophthalmological Society

President: Lester T. Jones, M.D., 624 Medical Arts Bldg., Portland 5, Oregon.
Secretary-Treasurer: John F. Tolan, M.D., 3419 47th Ave., N.E., Seattle 5, Wash.